SICK CHILDREN

DIAGNOSIS AND TREATMENT

DONALD PATERSON

SICK CHILDREN: DIAGNOSIS AND TREATMENT

SICK CHILDREN: DIAGNOSIS AND TREATMENT

A MANUAL FOR STUDENTS AND PRACTITIONERS

By

DONALD PATERSON

B.A.MANITOBA, M.D.EDIN., F.R.C.P.LOND.

Physician to Out-Patients, The Hospital for Sick Children, Great Ormond Street; Physician for Diseases of Children, Westminster Hospital

WITH 16 HALF-TONE PLATES AND 85 FIGURES IN THE TEXT

CASSELL AND COMPANY LTD
London, Toronto, Melbourne and Sydney
1930

First published - 1930

Printed in Great Britain

PREFACE

In this Manual I have attempted to deal with the commoner diseases of childhood as completely as the limits of space allowed, keeping constantly in view the needs of the senior student and the general practitioner. The reader, I hope, will find that the descriptions of symptoms, though concise, are adequate, nor have etiology and pathology been neglected; but, as the title suggests, the main stress has been laid on diagnosis and treatment as the surest way of making the book practical and helpful.

I have to thank Drs. Still, Cameron and Poynton for the loan of illustrations and for kindly help, and Dr. Robert Hutchison for the guidance I have found in his "Lectures on Diseases of Children" and "Food and the Principles of Dietetics," both published by Messrs. Edward Arnold & Co., to whom I am indebted for permission to quote two tables from Garrod, Batten, Thursfield and Paterson's "Diseases of Children." I must also thank the Editors of the *British Medical Journal*, the *Lancet*, the *Post-Graduate Medical Journal*, the *Archives of Diseases in Childhood*, and the *Practitioner* for permission to reproduce illustrations and papers of mine that have appeared in the pages of those publications. Nor must I fail to acknowledge the

courtesy of Messrs. Constable & Co., Ltd., in allowing me to copy some of the food tables and diet sheets that were compiled for Paterson and Forest Smith's "Modern Methods of Feeding in Infancy and Childhood," which deals with the subject of infant and child feeding much more at length than is requisite in a book on Sick Children.

Finally, I must thank my Secretary, Miss G. Frith, for her valuable help in the verification of references and the reading of the proofs.

D. P.

27, Devonshire Place, W.1.
April, 1930.

CONTENTS

CHAPTER		PAGE
1.	Normal and Abnormal Growth and Development	7
2.	Diseases and Injuries of the New-Born	36
3.	The Feeding of the Normal Infant and Child	62
4.	Diseases of Nutrition	96
5.	Diseases of the Eye, Ear, Nose and Throat	144
6.	Diseases of the Digestive System	155
7.	Diseases of the Respiratory System	191
8.	Diseases of the Circulatory System	225
9.	Blood Diseases	233
10.	Diseases of the Genito-Urinary System	251
11.	Diseases of the Nervous System	283
12.	Mental Defect	322
13.	Functional Nervous Diseases	352
14.	Diseases of the Bones and Joints	362
15.	Diseases of the Skin	372
16.	Infectious Diseases	389
17.	Tuberculosis	417
18.	Rheumatism	441
19.	Diabetes	462
20.	Intestinal Parasites (Worms)	472
21.	Syphilis in Childhood	478
	Appendix: Quantities of Certain Drugs which may be given to Children	488
	Index	489

LIST OF PLATES

PLATE		FACING PAGE
1.	Radiogram of wrist in a case of severe Rickets	104
2.	Radiogram in a case of Infantile Scurvy, showing a Subperiosteal Hæmorrhage at the lower end of the Femur	108
3.	Radiograms of Normal Infants immediately after Bottle-feed	142
4.	Radiogram of Cardiospasm	161
5.	Radiogram in a case of Lobar Pneumonia, taken after the Crisis	208
6.	Radiogram in same case as Plate 5, taken three days later	*Following Plate* 5
7.	Radiogram in same case as Plate 6, taken four days later	*Following Plate* 6
8.	Radiograms taken after Lipiodol Injection Fig. 1.—Normal Bronchial Tree in a case of slowly resolving Pneumonia. Fig. 2.—Extreme Degree of Tubular Bronchiectasis throughout whole of left lung.	216
9.	Radiogram showing Enlarged Thymus in an Infant ten weeks old	218
10.	Rash of Purpura	247
11.	Pyelogram in a case of Pyonephrosis	256

PLATE FACING PAGE

12. Brain of a Child ten months old, showing Tuberculous Masses 290

13. Brain in a case of Mental Defect and a Normal Brain 322

14. Pneumoradiograms showing Air in the Ventricles of the Brain 332

Fig. 1.—Lateral Ventricle.

Fig. 2.—Front View, showing both Lateral Ventricles.

15. Radiogram in a case of Osteogenesis Imperfecta 368

16. Radiogram showing Syphilitic Epiphysitis . 481

SICK CHILDREN: DIAGNOSIS AND TREATMENT

CHAPTER I

NORMAL AND ABNORMAL GROWTH AND DEVELOPMENT

Examination of a Normal Child

Much greater care is necessary in examining a child than is required for the examination of an adult. In the latter case great help has been given you by the patient's own description of his symptoms, but in the case of the child one has very often to depend on the observations of the mother or nurse, which may be quite helpful or exactly the reverse.

To make a thorough examination it is best to leave the child, especially if young, on the nurse's knee, keeping his attention concentrated on some object apart from yourself, observing him as closely as possible without allowing him to realize you are doing so. Avoid staring, especially at the child's face. If he becomes restive it is best to wait a moment until he has composed himself once more.

The head.—Note the shape of the head, and remember that at birth the *circumference of the head* should be 13 to 14 inches, at 6 months 17 inches, at 12 months 18 inches, at 18 months 18½ inches, at 2 years

18¾ inches, at 3 years 19¼ inches, at 4 years 19½ inches, at 5 years 20⅓ inches, at 10 years 21 inches and at 15 years 21¾ inches. If the head is markedly undersized, this is important, as it suggests microcephaly or failure of the brain to develop. If on the other hand it is definitely too large, this suggests some form of hydrocephalus.

The *shape of the head* is also of importance; in congenital syphilis the head is bossed and of the so-called "hot-cross-bun" shape, in hydrocephalus the forehead is overhanging, in rickets the head is large and square, and after certain forms of birth injury there is asymmetry of the head, the whole being skewed to one side or the other. One side of the head is larger than the other in cases of hemihypertrophy.

The fontanelle.—The fontanelle should close about the 18th month, but occasionally it closes or almost closes at an earlier age. Very early closure, especially in premature infants, suggests microcephaly. It may be widely open in cases of hydrocephalus, severe rickets or osteogenesis imperfecta, the child often reaching the age of 2 or 3 years before it closes.

The hair.—Normally the infant has a little soft down at birth, but occasionally there is a thick black brush or wig of hair. By 6 months there is a good growth of new hair. In rickets the hair becomes straight and dry, and in the cretin it is markedly so. In the mongol also the hair is straight, but softer. An examination of the scalp should always be made to exclude ringworm and patches of alopecia, also scurf and impetigo. In newborn babies swellings of the scalp at the site of presentation often appear. This is the *caput succedaneum* and is really a hæmatoma. Occasionally this becomes hard round the edge, suggesting a depressed fracture. This will be found, however, to be hardened clot only, and depressed fractures at such a site are extremely rare.

The eyes.—The *shape of the eyes* is important. Apart from such abnormalities as one eye blue and the other brown, congenital malformations should be looked

for, such as abnormalities in the shape of the iris and reflection back of the light from an opaque surface, suggesting cataract. In mongolism there is a *tendency to squint*, with an oval or slanting shape to the eyes and an exaggeration of the inner canthus. Other causes of squint are encephalitis lethargica, meningitis and brain tumours. Marked external squint may be present at birth or may be acquired as the result of some error of accommodation. Short lateral or vertical nystagmoid movements should be looked for, as these are significant in the diagnosis of cerebral lesions, and are occasionally found accompanying rickets in infants.

The normal infant should begin to follow a light at 3 or 4 weeks of age, and should definitely see objects by the time he is 2 months old. Failure to be attracted by, or to focus on a light after this age suggests *blindness*, which may be a local eye blindness or cerebral blindness due to mental defect. *Ptosis* may be congenital, in which case it is often familial; or acquired, due to some defect in the nerve supply to the eyelid; or the eye may be partially closed from œdema, due to some inflammatory condition. A tendency to squint seems normal in every new-born infant; this is of no significance and is quite transient.

The nose.—The *shape of the nose* is important. In congenital syphilis, with a chronic nasal discharge appearing shortly after birth, the bridge rapidly becomes infected and is destroyed, a permanent depression resulting. The pinched-in nose of the subject of "adenoids" or "nasal obstruction" is easily detected, and the undersized, ill-developed nose of the average asthmatic child is striking. In pneumonia the alæ nasi can be seen to work with each inspiration.

The ears.—Care should be taken to ascertain that the child can hear. If there is any question about his mentality being abnormal you should perform simple tests to see whether he hears sounds and how well. Find out whether coming into his room at night and making a noise wakes him, clap your hands loudly or drop something behind him, speak to him or whistle

to see whether he will turn towards these sounds. An examination of the drums should be made in every case as a routine, *especially in cases where there is fever without apparent cause*. Wax may obscure the examination and must be overcome by means of hydrogen peroxide and water, equal parts, put in several times daily, drying thoroughly with cotton-wool; or by syringing with a solution of sodium bicarbonate, one drachm to a glass of tepid water.

Complexion and face.—One thinks of the normal child as pink-cheeked and fair-skinned, but this will vary enormously with his parentage. The new-born infant is red-faced, then within a week or two his complexion becomes very pale and remains so until he is 2 or 3 months old at least, when a little colour begins to appear. Some children are always pale-faced, although not anæmic, and this is especially true of children with auburn hair. Black-haired families often have very white skins.

Tapping over the face in rickets, a mild spasm of the muscle occurs (Chvostek's sign). Extreme thinness of the face is a sign in lipodystrophia progressiva. In the extreme emaciation of the marasmic infant the sucking-pads protrude in the cheeks, the rest of the face being thin and pinched.

The mouth, lips and gums.—The lips are cracked and excoriated in congenital syphilis (rhagades), and they are dry and cracked in feverish conditions of all sorts. Herpes may appear about them as small blisters or crusts. The mouth is carp-shaped or turns down at the corners in mongolism, and is open widely, allowing the huge tongue to protrude, in cretinism. The lips may be pale and almost colourless in blood diseases, or dark-red or cyanosed in heart disease, asthma or pneumonia.

In scurvy the gums are swollen, red and hæmorrhagic. In ulcerative stomatitis the gums are so swollen that they may almost cover the teeth. Purpuric patches or hæmorrhages into the gums may be seen.

The throat.—In an examination of the child's mouth and throat it is best to wrap him in a blanket

or shawl, or else he should sit on his mother's knee. She should take both his hands in one of hers and hold his head firmly against her by placing her other hand over his forehead. In this way the tongue can be depressed with a teaspoon and, using a pocket torch, the mouth and throat can be explored in comfort. The number and condition of the teeth should be noted, and a glance should be sufficient to detect the presence of ulcers. Depressing the tongue brings the *tonsils* into view and the presence or absence of membranes or of tonsillitis can be noted.

The glands.—By palpating at the angle of the jaw the tonsillar glands can be felt if enlarged, and if the glands draining the adenoid region are enlarged they can be detected also by feeling behind the sternomastoid. Palpation of the glands in the axilla and inguinal region should take place as a routine, and also a careful examination of the abdomen, especially in the region of the mesentery, for tuberculous adenitis. Enlarged glands in this region suggest blood diseases such as leukæmia and Hodgkin's disease, or tuberculosis, syphilis or a new growth. Inflammatory conditions in the fingers or toes would of course give rise to adenitis in the axilla or groin.

The neck.—Palpation of the sternomastoid will often reveal a small hard lump, due to a hæmatoma into the sternomastoid at birth. Generally this disappears completely within the first year. There may be a tendency to wry-neck following this, or there may be a congenital shortening of the sternomastoid muscle, causing the head to be held to one side. In such cases the child should be placed on its pillow so as to press the head into the correct position ; in this way operation is often avoided. In *cerebellar tumour* the head is tilted toward one or the other shoulder. *Head retraction* is seen in cases of meningitis and other cerebral irritation, or there may be a stiffness of the neck only, as in poliomyelitis, spinal caries and inflamed neck-glands.

The chest and its shape.—The chest should be examined first by palpation, the ribs being felt for the

presence or absence of beading. Beading is most marked in cases of rickets at the costo-chondral junctions. Where there is a dislocation backward of the sternum, scurvy is suggested. In osteogenesis imperfecta there is a marked tendency to greenstick fracture of the ribs. If the chest be examined posteriorly the shape of the spine can be seen. Kyphosis suggests rickets if reducible, or spinal caries if irreducible. Scoliosis suggests congenital malformation of the vertebræ, or may be the result of poliomyelitis, rickets or long-continued general hypotonia. Pigeon chest results from rickets together with some form of respiratory obstruction or disability such as adenoids or broncho-pneumonia. In asthma the chest is barrel-shaped. Marked depression of the sternum is seen in thin, hypotonic children, who have had rickets and whose chests are sucked in along the line of the diaphragm (Harrison's sulcus). Depressions of the ribs or bulging of the chest on one side or the other suggests old rickets.

The lungs.—On listening to the chest the *breath-sounds* are noticed to be harsh, and inspiration can also be heard to some extent in addition to expiration. In the teething child who is salivating, sounds referred from the trachea are heard throughout the chest. Abnormalities in the chest of the infant and child are always of the grossest possible character, and slight changes are of much less significance than they are in the adult. *Percussion* should be light and not carried to any extent below the angle of the scapula, as the liver and kidneys are then readily palpated, giving rise to what may be considered abnormal dullness.

The heart.—*Percussion* of the heart is difficult and not very satisfactory. When gross abnormalities in size occur much can be done by palpating the apex-beat and feeling for the general cardiac impulse to the præcordia. On *auscultation* both the aortic and pulmonary sounds are best heard to the left of the sternum. The presence or absence of a thrill over the præcordia should be noted in every case, as it suggests mitral stenosis.

The abdomen.—In the infant the abdomen is normally full, and this fullness persists sometimes into the second year, but by the age of two the abdomen should be definitely flattened and no longer pendulous. Where the abdomen is *unduly large*, starch indigestion, cœliac disease and abdominal tuberculosis are some of the commoner conditions to be considered. The *condition of the navel* should always be inquired into, especially in the case of the new-born infant. Umbilical sepsis during the first week is of great significance and the cause of a long chain of symptoms, from jaundice to meningitis, pneumonia and general septicæmia.

On *palpating* the abdomen the liver should be felt for. Normally this extends one to two fingers' breadths below the costal margin. In cœliac disease the liver cannot be palpated. In liver cirrhosis it can be felt firm and hard down a finger's breadth only. Great enlargement of the liver is found in tuberculosis, new growth of the liver, catarrhal jaundice, and when it is displaced downwards by sub-diaphragmatic effusions or growths, or has fallen forward due to ptosis. The spleen is normally not palpable. When it can be palpated it suggests congenital syphilis, tuberculosis, new growth or blood diseases. Deep palpation of the abdomen for enlarged kidneys or other masses should be carried out. The child should be examined in a good light, standing, if possible, to allow herniæ to be seen, and the presence or absence of the testicles in the scrotum should be ascertained by palpation. Phimosis should be looked for, and pin-point meatus or meatal ulcer in cases where circumcision has been done.

The legs and arms.—The *shape* of the legs and arms should be noted. *Knock-knee* or *bow-leg* suggests rickets. *Abnormal shortness* of the arms and legs is present in achondroplasia. Walking with a *waddling gait* suggests achondroplasia or congenital dislocation of the hip on one or both sides. *Limping* may be seen in simple trauma of the foot, leg or hip or in tuberculous disease. *Pain* on movement of the limbs in infants is suggestive of scurvy. A *rolling staggering gait* suggests

cerebellar tumour, post-diphtheritic paralysis or lesions of the nervous system.

The knee-jerks.—An examination of the knee-jerks is of the greatest importance. In cerebral lesions such as spastic diplegia the knee-jerks are much increased and there is a spasm or spasticity of the lower limbs. *Absence* of the knee-jerks is seen in poliomyelitis and Friedreich's ataxia. Whether Babinski's reflex is plantar or flexor is of less importance in infants. It tends to be extensor normally. Ataxia of the arms is shown in the nose-finger test, both in cerebellar and cerebral lesions.

Development of the Special Senses, Speech and Motor Control

Shortly after birth the child should follow a bright light with his eyes. Certainly by one month a normal child will follow a bright object, and will recognize his mother or nurse between the ages of 2 and 3 months. Objects placed in the hand can be gripped tightly from birth onward, but the child is not able to reach out and grasp objects before 3 to 4 months of age. Things are held properly in the hands and passed from one hand to the other at the age of 6 to 9 months, and a normal infant of 15 to 18 months learns to feed himself with a spoon.

Speech is acquired slowly. By one year most infants are able to say "Ta" for "Thank you," "Mama" and "Dada," and can imitate the sounds of various animals. Single words are clearly spoken by 15 months, and short two- or three-word sentences by 2 years, the child having acquired quite a large vocabulary by this time. Variations in the rate of learning to speak are very great, some children articulating clearly at a year and forming long sentences by the end of the second year, while others are much more backward. Such variations in speech do not in any way signify mental abnormality. Small nursery rhymes can be learnt by 2½ to 3 years, and the child is able to say his letters by 4 or 5 years. By the age of 5 to 6 years he can learn to write.

Walking.—The average child should sit up by 6 months, pull himself up in his cot into an upright position by 9 or 10 months, and walk at 12 or 14 months. He should walk quite well by 18 to 20 months. Overfat children acquire these capacities much more slowly. This may be due only to their weight, or in part to flabbiness, or even to rickets. There is a feeling among the public, especially among old-fashioned nannies, that a child should not be allowed to sit up, stand or walk, even when he shows marked inclination to do this himself. Experience is directly contrary to this belief, and nothing but good can come from early progress in this direction.

Training of the Child in Good Habits of Micturition and Defæcation

The infant should be placed on his chamber at regular intervals after each feed almost from birth, and an association should be firmly established in the child's mind between the feel of the chamber, together with the position adopted, and the act of micturition or defæcation. Gradually this association will become so fixed in his mind that the bladder and bowel are only emptied when he is placed on his chamber. As the feeds are spaced farther and farther apart and become less and less fluid, control becomes more easily acquired and more perfect. Even although the infant may pass his water between feeds he should nevertheless be held out at the set times, until the bladder is again emptied. Much time should be devoted to this in the early months. A quiet insistence will greatly repay the mother or nurse.

Except for occasional lapses, the normal child may be kept dry during the day, between the ages of 9 and 12 months, and he should certainly be dry at night between a year and 18 months. Variations in the age when the bladder is controlled are common, however. Exacerbations of wetting occur at periods when dentition is active or in extremely cold weather.

A few minims of tincture of belladonna at such times will aid the child in acquiring and keeping control. By 2½ years every normal child should be able to ask to be attended to during the day, and this should be on waking, after breakfast, at dinner, at tea and at bedtime only. During the night some children require to be lifted up at 10 p.m., but this should be discontinued as soon as possible. Giving the last meal at 5 p.m. and no drinks afterwards is a very great help in getting such children to remain dry during the night period.

Defæcation.—The child can usually be trained to pass his motions into the chamber at stated times when from 2 to 3 months old. If he is constipated, paraffin emulsion or some mild aperient may be necessary, but in the ordinary way if there is difficulty a little gentle stimulation of the anus with a well-vaselined finger-tip or the tip of a soft rubber catheter will produce defæcation. At 9 or 10 months the child should be able to sit and balance on his chamber and should be left there after breakfast and after tea for the purpose of emptying the bowel.

Proper Clothing

The clothing of the infant and child naturally varies with the climate and the customs of the people. Among the well-to-do where the houses are properly heated, less indoor clothing and more outdoor clothing is required than among the poor, who need to wear more indoor clothing. Undoubtedly the children of the hospital class are as a rule enclosed in too many garments of an inferior quality, seven or eight layers of shoddy cotton being of less value to the child than one or two woollen garments.

The infant requires a long-sleeved woollen undervest and a long flannel petticoat which can be folded over the feet and legs. Socks should be worn. A cotton or linen gown is usually worn outside and during the winter months a woollen jacket as well. Shortening of the clothes should take place not depending on the age

of the child, but on the progress he is making and the weight he acquires. A large infant of 10 lb. may be safely shortened into woollen knickers and jersey when one or two months old, while in small premature infants this cannot take place until they are about 5 or 6 months old. It is a great advantage to the infant to be free to kick and develop his limbs, and when in doubt it is always best to do away with the encumbrance of the long clothes. The infant binder should be discontinued at the end of a fortnight, a knitted abdominal belt being substituted, and this in its turn discontinued after one to two months. Too often the binder "works up" to the child's thorax, compressing it and being of no practical value.

Between the ages of one and two years summer- or winter-weight combinations or woollen vest and pants should be worn next the skin ("Chilprufe" is a good make). Woollen knickers and jersey during the winter, cotton suits during the summer, are most suitable. In very warm weather silk or cotton combinations may take the place of the woollen ones, but in climates where the temperature drops towards evening the greatest care must be taken in abandoning the wool.

Caps and bonnets.—The infant born in the summer need not wear a bonnet or cap when taken out, but those born during the winter months require some protection until the hair has grown sufficiently. All children require woollen caps or some form of head covering during severe weather.

Shoes.—At the age of one year, when the infant commences to walk, it is best to provide him with shoes having firm soles and small heels. A good make is the "Start-Rite" shoe, but there are various other makes which are equally satisfactory. The heels should be narrow enough to fit snugly, whereas the toe of the shoe should be broad, allowing plenty of room. If there is even the slightest tendency to flat foot from laxity of muscles or overweight, small instep supports should be inserted, or the heels wedged $\frac{1}{8}$ inch to $\frac{3}{16}$ inch on the inner side, passing away to nothing on the outer side,

like a wedge of cheese. At least two pairs of shoes should be kept for each child, so that the feet can be rested.

Weight of the Normal Child

An average infant weighs at birth about 7 lb. There is a loss during the first four to seven days of anything up to half a pound, but this is quickly regained and by the tenth to twelfth day the weight is once more normal. Thereafter a gain of from 6 to 8 ounces a week occurs for the first five months, and of about 4 ounces per week from five months to one year. At five months the infant has doubled its weight and at one year it has trebled it. In private practice much greater gains than these are found. It is not in the least uncommon for a 7-lb. infant to weigh 18 lb. at 6 months and 25 lb. at one year. This does not necessarily mean that the child is more healthy, but very often it has thrived well due to the extremely good care bestowed on it. The *weekly weighing* of infants ought to be insisted upon, as it is only by weighing that we can gauge the progress that is being made. For the first six months the infant ought to be weighed weekly, from six months to one year fortnightly, from one to two years monthly, and from thence up to 12 years every two or three months. No well-cared-for healthy infant should lose weight. The weight may tend to increase more rapidly one week than another, but there should always be a gain. Cutting teeth, a change of nurse or human environment, vaccination, may all cause stationary weight temporarily. In *older children* emaciation or wasting may occur from too great activity and nervous exhaustion, dental caries with chronic indigestion, from rheumatism and tuberculosis, diabetes, etc. The constitutionally over-active child is extremely difficult to fatten and is a problem in practice. This is dealt with under "Nervous Exhaustion" (p. 352). Older children gain weight at irregular rates.[1] It has been found that some children gain in the summer only

[1] Emmett Holt, "Observations on the Health and Growth of Children in an Institution," *Am. Jour. Dis. Child.*, Vol. 1, July 1923, pp. 1-22.

and are stationary or gain very little in the winter, and vice versa. The weight of normal children at various ages is shown in Table I.

Growth in Weight

TABLE I

Weight of a Normal Child from Birth to Fifteen Years

Age.	Boys. Weight in lb.	Girls. Weight in lb.
Birth	7½	7
6 months	16	15½
1 year	21	20
2 years	27	26
3 „	31	30
4 „	35	34
5 „	41	40
6 „	45	44
7 „	50	48
8 „	55	53
9 „	60	58
10 „	67	64
11 „	72	70
12 „	80	82
13 „	88	92
14 „	99	101
15 „	111	109

Obesity

It is extremely difficult to explain all the causes of obesity. After encephalitis lethargica some children become extremely obese, and it is thought that there is a lesion in such cases in the region of the pituitary or corpora quadrigemina. After postbasic meningitis with hydrocephalus undue obesity has been noted, and this is also the case with a pituitary tumour. Some Jewish children at the age of 8 to 12 tend to become extremely obese and to develop a picture very like Fröhlich's syndrome. This seems to be a functional rather than an organic condition. It would appear as if the body were able to absorb and store up fat, but

unable to call on it again and in this way to reduce the obesity. With this one-way valve action, when the fat pours in and is not burnt up, some children are able to subsist on an extremely small intake. Actually, however, a vicious circle is formed. Fat is laid on, probably due to some metabolic error. Once having become fat, the child is less active, and, being less active, tends to put on more fat. This vicious circle continues. With the increased weight of the child there is a tendency for the arches of the feet to break down, and with sore feet even less inclination to activity results.

Distribution of fat.—In the male child there is a tendency to a feminine type of distribution, especially of the breasts. In the female much fat is laid on over the hips and shoulders. In about one-third of the cases there has been obesity from birth, in one-third it occurs between the ages of 4 and 7 and in the remainder the onset is between 10 and 12 years of age. It is slightly more common in females than males. There is not only an increase in weight, but the whole child is overgrown, being taller and broader than the normal for that age. There has been a definitely accelerated growth. As adults these children may not be abnormally large, in fact many of them are under average size. The result of this accelerated growth is that there is premature fusion of the epiphyses as shown by X-rays. Although the sexual characteristics are said to be retarded, they are probably normal for the age of the child, though not for the physical development.

Treatment.—A careful history should be taken *to exclude organic disease* suggesting encephalitis, meningitis, disease of the thyroid, or pituitary insufficiency.

The *diet* should be gone into and the fat and carbohydrate reduced to a minimum. Very few eggs and very little milk should be given, and there should be a great reduction in bread, cereals, potato, etc. The child should have a full allowance of meat, spinach, cabbage and lettuce. Sugar should be reduced to a minimum. Only three meals in the day should be allowed. Drinks of water need not be restricted.

Fruit of all sorts may be given in moderate quantities in place of milk puddings.

Diet for overweight children.—At breakfast raw fruit, such as orange or grapefruit or an apple, should start the meal. This is to be preferred to cereals, which have a high caloric value and are fattening. As a second course, cold tongue or the lean portion of bacon or ham, or a liberal helping of fish should follow. Regarding eggs, the white is permissible, but an occasional yolk only should be given, say two in the week at most, as it has a very high fat-content. The drinks should consist of freshly brewed tea or plain water. Milk should be avoided or taken in small quantities only. Toast and bread and biscuits have all the same food value and are extremely fattening. One small slice of toast should be sufficient. Bread which is free of carbohydrate, such as that supplied by Callards, 35 New Cavendish Street, London, W.1, is sometimes helpful in completing the meal.

For dinner the child should have a liberal supply of protein or meat, and in addition as much green vegetables, such as spinach or sprouts, as he will take. Other vegetables such as potato and cauliflower must be taken in small quantities only. Carrots, turnips, parsnips, peas and beans can be given in moderate quantities, say a heaped tablespoonful. Milk or steamed puddings should be avoided, and in their place fresh or stewed fruit may be given as a second course, with water to drink.

At tea-time a cup of weak tea, with a small biscuit or a meat or fish-paste sandwich, should be sufficient. For supper, soup, except potato- or pea-soup, is suitable. For the older child salads may be given at this meal and at any other meal, as they are satisfying and not fattening. Plasmon preparations will be found most helpful at this meal.

Sweets of all kinds and chocolate should be excluded from the child's diet, and little sugar should be allowed.

Massage, cold baths and *vigorous exercise* of all sorts should be persevered with.

With regard to *medicinal treatment*, ½ grain of thyroid gland twice daily may be safely given, and if the child is under close medical observation this should be slowly increased up to 1½ or 2 grains a day. Symptoms of tachycardia and flushing should be watched for. Pituitary gland, ½ grain twice daily, increased slowly to 2 grains, has been advocated by some workers.[1, 2]

Fig. 1.—Case of obesity aged 11 years.

Prognosis.—Those cases in which no organic lesion or preceding disease can be ascertained, and which are assumed to be functional, become normal late in adolescence. (Fig. 1.)

[1] H. Gardiner Hill, I. Jones and J. Forest Smith, "Pituitary Obesity in Adolescence," *Quart. Jour. Med.*, 1925, pp. 309-326.

[2] F. S. Langmead and E. G. B. Calvert, "Obesity in Children," *Lancet*, Nov. 29, 1924, pp. 1111-1115.

Thyroid Enlargement (Goitre)

True exophthalmic goitre (Graves's disease) is a great rarity in childhood, in fact it is practically unknown. Occasionally one sees an enlargement of the thyroid, benign in character, in very young children, and this as a rule gives rise to no symptoms. The author has once seen a case which gave rise to typical cretinism at the age of four (acquired cretinism). Preceding puberty, especially in girls, an enlargement of the thyroid is extremely common. This may be accompanied by a certain amount of tachycardia and flushing, and persist at each period until the period of puberty has been passed.

Fig. 2.—Enlarged thyroid in a child showing no symptoms.

Five minims of the French tincture of iodine or 3 minims of ordinary tincture of iodine in a little milk three times daily is of value in such cases, and should be given for a month, then stopped for a fortnight, and so on. (Figs. 2, 3.)

The Thin Child

Some children have been well nourished and then progressively lose weight, becoming thinner and thinner. Other children have always been thin and over-active, coming of a thin stock.

The thin child must be carefully examined to exclude

organic disease as a cause of wasting. Diabetes mellitus, interstitial nephritis, congenital syphilis, rheumatism, tuberculosis and chronic fat and starch dyspepsia, may all produce progressive wasting, but a careful examination will exclude such diseases. A very rare condition,

Fig. 3.—Goitre in a child who later became a typical cretin. On treatment with thyroid the child became normal, but the enlarged thyroid persisted.

progressive lipodystrophia, may occasionally be met with. As a rule it is the upper half of the child that is involved in this condition, and there is an almost entire absence of the subcutaneous fat, the face, arms, chest, etc., presenting a picture of extreme emaciation.

There still remains the commonest type of thin child, the **constitutionally thin child (nervous-exhaus-**

tion child). There is an ever-increasing number of these thin children, who are the children of thin, over-active, intelligent parents. The mother's complaint is that she cannot get any flesh on him; he has always been thin, although possibly quite a plump infant.

Etiology.—Such a patient has a defective power of assimilating food, especially fat, and tends from his nature to be over-active and to expend too much energy daily. The result of this is that the balance between the intake of food and the output of energy is too nearly equal. An attempt to increase the intake by the addition of cream, milk, butter and eggs produces a bilious attack. These children then suffer frequently from gastric or acidosis attacks (*see* Chronic Fat and Starch Dyspepsia, Cyclical Vomiting, etc., Chapter IV).

Treatment.—The problem reduces itself to that of *cutting down the child's output of energy* to the point where with a moderate intake of food he has a balance on the right side, so that he thrives and gains weight. This can be done by limiting the length of his day and arranging for a *midday rest*. Little children of this type up to the age of three or four require to be off their legs for half the day. *School* or *kindergarten* should be started early, in order to enforce rest and discipline. There is no reasonable doubt that carbohydrate, especially in the form of sugar, is extremely well tolerated by such children, and powdered glucose or plain sugar is a useful adjunct to their diet.

Growth in Height

The normal child.—It will be noted that the normal child at birth is from 18 to 22 inches in length, and by one year he is 28 to 30 inches in length. From this time on there is a growth of roughly 2½ inches per year. Up to the age of 12 the girls are slightly shorter than the boys, at 12 and 13 they are slightly taller, and from 14 years on the boy once more leads in height.

Growth in height, like growth in weight, is not a steady process. We tend to go ahead by leaps and bounds, nor can growth be said to be definitely

seasonal. It is considered by some that being confined to bed causes more rapid growth, but this is not always the case. The height of normal children at various ages is shown in Table II.

Various factors operate in retarding a child's growth and producing infantilism, dwarfism or stunting. On the other hand gigantism is also seen in childhood.

TABLE II

HEIGHT OF A NORMAL CHILD FROM BIRTH TO FIFTEEN YEARS

Age.	Boys. Height in in.	Girls. Height in in.
Birth	21	21
6 months	25	25
1 year	29	29
2 years	32	32
3 „	35	35
4 „	38	38
5 „	42	41
6 „	44	43
7 „	46	46
8 „	48	48
9 „	50	50
10 „	52	52
11 „	54	54
12 „	56	57
13 „	58	59
14 „	61	60
15 „	64	62

Infantilism.—Any *gross organic lesion* in some vital organ will produce infantilism. Thus congenital heart disease, congenital cystic kidneys and interstitial nephritis, congenital atelectasis of the lung, cœliac disease, all retard the growth and development of the child. These will be dealt with separately and nothing more need be said here. Those cases of retarded growth and development due to *endocrine insufficiency* possess one very clear example in the cretin. *Congenital absence of the thyroid*, or congenital thyroid insufficiency, is

common in childhood. In severe *rickets*, especially that type of rickets accompanying interstitial nephritis and known as renal dwarfism, there is gross stunting with bony deformity as a rule.

Gigantism or overgrowth : precocious puberty.—Gross over-development or overgrowth is uncommon. With tumours of the suprarenal a type of overgrown, over-developed child, with florid red cheeks, has been described by Guthrie and labelled " brewer's drayman." Ovarian and suprarenal tumours account for some of the cases of precocious menstruation seen in children of 3 years and upwards.[1]

Sleep

The normal child.—The following table shows the amount of sleep required by children at various ages :

TABLE III

Birth	20–24 hours
Six months	18 ,,
One year	14–16 ,,
Two years	12–14 ,,
Five ,,	10–12 ,,
Ten ,,	10–11 ,,
Sixteen ,,	9 ,,

In children up to the age of four or five it is well to introduce a midday rest. Up to the age of four the rest is best given before dinner, say from 11 to 1, but between four and five this is best given after dinner, from 1.30 until 3.

Abnormalities in sleep.—In ordinary healthy infants there is a vast difference in the amount of sleep required, some children requiring much more than others. Children who are played with and become *too excited* or who are allowed to be *on their legs too much* sleep badly. *Digestive disturbances, too late supper, overfeeding the last thing at night,* tend to produce night terrors and disturbed sleep. The *presence of tonsils and adenoids* blocking the child's airway will produce broken sleep. At intervals throughout the night slow

[1] E. A. Cockayne, in Garrod, Batten, Thursfield and Paterson's " Diseases of Children " (Edward Arnold & Co., 1929), p. 509.

asphyxia occurs, with sudden bursts of crying in which the child recovers his oxygen. In infants *hunger pains and wind colic* are often the cause of disturbed sleep. Many infants waken each time *water is passed* throughout the night and refuse to settle off again unless changed. As they get older and micturition is less frequent the nights become better and better. With *teething* there would appear to be definite *neuralgia* in some cases, and this undoubtedly wakens some children. Accompanying dentition there is as a rule *drooling* or hypersecretion of saliva. At night, especially when the child is lying on its back, the saliva tends to collect in the pharynx and larynx, causing intermittent bursts of *coughing* and often waking the child.

In *encephalitis lethargica* sleep by day and wakefulness at night is common. In a child who has been perfectly normal and who after some febrile illness develops restlessness and wakefulness at night, with a tendency to sleep during the daytime, encephalitis lethargica may be suspected. *Tumours of the brain*, especially *pineal tumours*, are said to cause increased drowsiness, but these are very rare. In most febrile illnesses drowsiness occurs.

Fresh Air, Sunshine and Exercise

Fresh air.—A London child requires from four to six hours' fresh air every day as a minimum for good health. Up to the age of four or five he should be out both morning and afternoon.

Sunshine (artificial and natural).—Natural sunshine is such a rarity in some quarters of the year that the question of *artificial sunshine* has seriously arisen. On the whole, however, the value of this is over-estimated, and it is not necessary for a normal, healthy child, provided that small doses of cod-liver oil are given daily. On the other hand it is useful in the treatment of rickets and in raising the immunity of those children who are prone to catarrhs and those who suffer from flabbiness and watery tissues. Children with asthma also benefit by artificial sunshine.

Undoubtedly after influenza and colds during the winter months a course of artificial sunshine is of very definite value to most children, if well administered.

Sun-baths.—During the summer months whenever possible the child should be stripped and allowed to run and play with the sun shining directly on the body. This, combined with sea air, is of the utmost value to every normal child and especially to those who have been debilitated by rheumatism or tuberculosis. Exposure to the natural sunlight, just as to artificial sunlight, should be gradual after the child has had a severe illness.

Exercise.—While many children suffer from confinement and lack of fresh air, very few children, once they are able to walk, suffer from lack of exercise. They manage to take the maximum amount which is good for them, whether allowed out or kept indoors. On the whole little children tend to be too active and to take more exercise than is beneficial.

Normal Dentition

First set of teeth or primary dentition.—The date of the eruption of the different teeth varies, but on the whole the teeth are cut in the following order and about the following times :

1. Lower central incisors, 5 to 10 months.
2. Upper central and lateral incisors, 8 to 12 months.
3. Lower lateral incisors and lower and upper first molars, 12 to 14 months.
4. Lower and upper canines, 16 to 22 months.
5. Lower and upper second molars, 24 to 30 months.

Delayed dentition.—As a rule the teeth appear in pairs. Many abnormalities in the appearance of the teeth and the type of teeth cut will appear to be hereditary, and there can be no doubt that good teeth run in families.

Probably one of the chief factors in causing delayed and imperfect dentition is the presence of *rickets*. This disease is due to a lack of vitamins, fresh air,

sunshine and proper food, all of which contribute to cause defective calcification of the bones, including the teeth. This delays both the date of eruption and the structure of the teeth. The giving of suitable doses of cod-liver oil, a well-balanced diet,[1] plenty of fresh air and sunshine, will guarantee as far as possible normal times of dentition and normal teeth.

Symptoms of dentition.—Two extreme theories of dentition are advanced: (1) That there are myriads of diseases and upsets definitely due to cutting the teeth; (2) that teething is responsible for nothing but the cutting of teeth.

Those who are fortunate enough to have infants under their charge in a hospital ward during the period of dentition, when the temperature, pulse and weight are closely observed, are able to form an excellent opinion of dentition. It would appear that, varying with the individual, this physiological process may become pathological, just as is the case with childbirth.

Fever.—Some children are inclined to be fretful and out of sorts, and actually have a temperature immediately preceding the cutting of a tooth, without any other ascertainable cause of the fever.

Vomiting.—During the eruption of the first set of teeth, especially between the ages of one year and 18 months, when an attempt is being made to introduce more solid food, there is often a strong tendency to vomit any solid constituent of the diet. This tendency rights itself rapidly after the eruption of each tooth, and is probably mechanical only. Because of the tender gums the child refuses to masticate the solid particles and swallows them in that form.

Diarrhœa.—When diarrhœa occurs during dentition the stools are characteristic; they are loose, but of a good colour, and do not contain undigested particles. In addition, the infant does not fail to gain weight. Dietetic measures to check the diarrhœa, such as reducing the feed or changing the child to some other food, do not check the frequency.

[1] Mrs. Mellanby, "Diet and the Teeth," *Brit. Dent. Journ.*, July 1927.

Neuralgia.—It seems reasonable to believe that at this time dentition is accompanied by pains of a neuralgic character. The face may become flushed on one or both sides, one side being warmer than the other. Very often the infant bangs its head and apparently gets some relief. The hand is held to the side of the face, and the sleep is restless and disturbed.

Cough.—Children may get bronchitis at the same time as dentition, but the common cough accompanying dentition is not bronchitic in the majority of cases. The saliva which normally flows from the mouth during the day flows to the back of the pharynx at night, where it tends to occlude the larynx. Thus the child coughs at intervals, especially during the night hours when placed on the back.

Convulsions.—In a normal child convulsions never accompany dentition. On the other hand in a rachitic child or mentally defective infant during the period of dentition convulsions may occur.

Eczema and skin eruptions.—Infants prone to eczema have acute exacerbations accompanying the cutting of each tooth, the eczema clearing up entirely when dentition is complete. Lichen urticaria and various other urticarial rashes appear and disappear at this time also.

Treatment of the child while teething.—Since rachitic infants have more difficulty in cutting their teeth than non-rachitic children, we should first make sure that the diet is a well-balanced one, and in addition that the other anti-rachitic factors are present, including cod-liver oil in some form. A routine purge for a few days will be found most useful, especially grey powder given at bedtime, with a little milk of magnesia first thing the following morning. If the disturbance at night is severe, causing much loss of sleep, chloral hydrate is recommended.

Great care must be taken that before making a diagnosis of teething all organic disease has been excluded. Pink disease is most often put down to teething. (For the other symptoms of this disease *see* p. 309.)

Secondary dentition (permanent teeth):

First molars appear	between	the	ages	of	5 and 7.
Central incisors	,,	,,	,,	6½ ,,	8.
Lateral incisors	,,	,,	,,	7 ,,	9.
First bicuspids	,,	,,	,,	9 ,,	11.
Second bicuspids	,,	,,	,,	10 ,,	12.
Cuspids	,,	,,	,,	11 ,,	14.
Second molars	,,	,,	,,	11 ,,	13.
Third molars	,,	,,	,,	16 ,,	21 or later.

Heredity

It is commonly thought, and taught by some, that heredity plays a very small part in a child's physical or mental make-up. There can be no doubt that this is a great error, and to the pædiatrician it is a commonplace to see the physical characteristics of one or other parent completely reproduced in their child. This applies to the colour of the hair or eyes, the general build of the body, stocky or slim, the expression, and even to the features, beautiful or plain. As regards the mentality, it is argued that bright or dull, witty or stupid children are so because of association with people of such a disposition. Again, the author cannot agree. Some children are born with a temperament which they have clearly inherited, and they take life seriously or otherwise on this account. The author feels extremely fatalistic as regards a fundamental change in any of these inherited characteristics. The child who might be weakly and good soil for disease may be kept strong and healthy and physically improved by his parents, because of their better understanding of this. Again a child may have inherited weakness of character, but by steady training may develop strength in this direction, so that he becomes a first-class member of society. Nevertheless, such a child, under stress, may demonstrate all his inherited weaknesses.

In the adoption of children, which is now practised on a large scale, the greatest care should be exercised to exclude children who may have inherited undesirable tendencies. The family history should be carefully inquired into for asthma, epilepsy and mental defect, and in addition a Wassermann test should be

done to exclude syphilis. The mentality and appearance of the parents should also be ascertained, as almost all their chief characteristics may later be reflected in the child.

Most of the definitely hereditary diseases, such as hæmophilia and pseudohypertrophic paralysis, are of rare occurrence. It is well known that a predisposition to disease is much more often inherited than the disease itself.

Just as in farm breeding the greatest care is taken to obtain the best stock and it is expected in this way to get the best results, so in human life the children of strong, healthy, well-balanced parents should and do possess the best physical and mental make-up.

Mortality in Childhood

The following table, taken from the Annual Report of the Chief Medical Officer of the Board of Education, is of interest to School Medical Officers. It shows the principal causes of death among children of school age (between 5 and 15) in England and Wales in the year 1928.

TABLE IV

Disease.	Number of deaths.	Per cent. on total deaths.
1. Tuberculosis	2,173	16.4
2. Violence	1,607	12.1
3. Diphtheria	1,565	11.8
4. Bronchitis, pneumonia, and other respiratory diseases	1,526	11.5
5. Meningitis and nervous system ..	1,116	8.4
6. Heart disease	822	6.2
7. Measles and whooping-cough ..	554	4.2
8. Rheumatic fever	513	3.9
9. Appendicitis	472	3.6
10. Acute and chronic nephritis ..	276	2.1
11. Acute infective osteomyelitis ..	255	1.9
12. Influenza	237	1.8
13. Scarlet fever	231	1.7
14. Tonsillitis, etc., adenoid vegetations	200	1.5
15. Other conditions	1,733	12.9
	13,280	100.0

From the table it will be seen that *tuberculosis* heads the list. No disease is more preventable, however, than tuberculosis in childhood (*see* p. 425 for methods of prevention). *Violence and accidents* come second. *Diphtheria* is third, and both the prevention and treatment of this lie entirely in the hands of the medical profession, if the parents are willing (see p. 405). Deaths from diphtheria should be entirely avoided if the population is properly immunized. The mortality from *surgical diseases*, such as appendicitis, osteomyelitis, and tonsils and adenoids, can be considerably reduced with earlier treatment, surgical aid being sought much too late as a rule. We now have prophylactic treatment for *measles*, although it cannot be claimed to be highly efficient. Our treatment for *whooping-cough* remains ineffective. Nevertheless, by taking great care in infancy and early childhood to keep the child away from infection, these two complaints have become commoner in older children, with a consequent great reduction of the mortality (*see* pp. 396, 400). *Nephritis* is very often the result of nasopharyngeal infections. Earlier and more efficient removal of septic tonsils in the future is bound to reduce the incidence of this disease (*see* p. 271). *Rheumatic fever and heart disease* (excluding congenital heart disease) are very much in the eye of the profession at the moment, and some efficient treatment is being strenuously sought for. Prophylaxis against this disease lies largely in social reform and keeping children well, but the same could be said of almost every disease mentioned in the table.

The mortality in childhood generally must be regarded as comparatively satisfactory, however, as it shows a steady drop, especially since the War. This may be attributed in part to additional knowledge of disease and its more efficient treatment, but a very important factor is undoubtedly the improved social condition of the poor.

Table V shows the remarkable drop which has taken place in infant mortality during the last half-century. It is taken from the Registrar-General's Statistical

Review of England and Wales for 1927 (H.M. Stationery Office).

TABLE V

INFANT MORTALITY (ENGLAND AND WALES), DISTINGUISHING MORTALITY FROM DIARRHŒAL DISEASES

(*Deaths under one year of age per* 1,000 *live births.*)

Year	Diarrhœal Diseases	Other Causes	All Causes
1861–65	15	136	151
1881–85	14	125	139
1901–05	23	115	138
1911–15	19	90	119
1916–20	9	81	90
1921–25	8	68	76
1925	7	68	75
1926	8	62	70
1927	6	64	70

A standard which can be usefully applied to present conditions is furnished by the infant mortality from all causes for New Zealand during the period 1921–24, viz. 43.4 per thousand.

CHAPTER II

DISEASES AND INJURIES OF THE NEW-BORN

Umbilical Infection (Sepsis Neonatorum)

Etiology and pathology.—It is impossible to estimate the frequency of umbilical infection, but there seems little doubt that it is much more frequent than is generally supposed. Sepsis often occurs from faulty technique after the birth of the child, the bath being commenced too soon, or the dressings on the cord being allowed to become infected. The infection may remain in the stump of the cord, causing a localized discharge from an area of fleshy vegetation which fails to dry up for some weeks. On the other hand it may pass up the obliterated vessels to the liver, where it either infects this organ or more frequently causes a general septicæmia. The streptococcus is by far the commonest organism found.

Symptoms.—If the obliterated vessels are involved, they can often be felt cord-like beneath the skin of the abdomen. If there is a general septicæmia, however, which is more common, the child is extremely ill from the start. A variety of clinical pictures may now arise, depending on where the brunt of the infection has fallen.

Acute septic jaundice.—Acute hepatitis may result, with fever, extreme jaundice, drowsiness and wasting. The child sucks badly and in many cases the condition is fatal. The author has, however, seen occasional cases in which the patients survive, after having been given antistreptococcal serum and transfused, with much careful nursing. In one such case cirrhosis of the

liver resulted and mental defect, but others seem to recover completely.

Diarrhœa accompanying septicæmia.—This is most often fatal. Breast-milk should be persevered with in such cases and general treatment for septicæmia resorted to.

Skin infections, such as exfoliative dermatitis, also occur as a result of umbilical sepsis.

The relationship between such a general septicæmia and *melæna neonatorum* has not been clearly defined. *Pneumonia* in new-born infants is undoubtedly the result of septicæmia, and *peritonitis, empyema, pericarditis* or *meningitis* may occur, though these are rare.

General treatment.—The *nursing* is of the first importance. Undoubtedly if the child can be kept going with suitable food, there will be complete recovery in a considerable number of cases. As a rule the infant is under the care of a midwife or maternity nurse, with no help from the mother, and the result is that this overworked attendant fails to make the best of the case. Records of the infant's intake of food should be kept to ensure that a sufficient quantity of nourishment is being given. If breast-fed, this means that careful test-feeds must be given.

To gain the upper hand of the general infection, *blood transfusion* or antistreptococcal serum seems of very definite value. 10 c.c. of the parent's blood per pound of the infant's body-weight should be given intravenously, or 20 c.c. of antistreptococcal serum may be given subcutaneously.

Icterus Neonatorum

True icterus neonatorum must not be confused with the other forms of jaundice which may commence at birth. By icterus neonatorum is meant simple jaundice, coming on in from two to five days after birth, lasting up to a week, causing few or no symptoms and requiring no treatment.

Etiology.—The cause of true icterus neonatorum is still unknown. With the van den Bergh reaction

the jaundice can be shown to be hæmatogenous in origin. Icterus is much less common in private than in hospital patients, suggesting that with care and the better condition of the patient at birth it is less likely to occur. On this account slight sepsis or chills are regarded by some as the cause.

Differential diagnosis.—Care must be taken to differentiate between true icterus and *gross jaundice due to umbilical infection* with a secondary acute hepatitis. In such cases the child is extremely ill. *Syphilitic cirrhosis of the liver* must also be excluded, and finally *congenital obliteration of the bile-ducts* (*see* p. 51) must be considered, where stools remain light in colour and jaundice may be delayed.

Tetanus Neonatorum (Trismus Neonatorum or Lockjaw)

Etiology and pathology.—This disease is much less common now than formerly. It was shown at one time to be due to an infection of the umbilicus by the tetanus bacillus, the toxins acting at a distance on the brain and spinal cord. Inoculation of scrapings from the umbilical cord into guinea-pigs has produced the disease. Some authors hold that the same clinical picture can be produced by other organisms, such as the streptococcus, by producing a meningitis, from which the patient may recover.[1]

Clinical picture.—On the third to fifth day the child shows a tendency to sudden spasm. The hands and jaws are clenched, and any attempt to force the jaws apart results in a sudden spasm of the whole infant. The nourishing of such an infant is a great difficulty. Spasms interfere with its respiration, and death, if it occurs, is due to general exhaustion.

Prognosis and treatment.—The majority of the cases are fatal. Occasionally, however, if they are treated early and given antitetanic serum, both subcutaneously and intrathecally, recovery takes place. Relatively a large dose of the serum is required.

[1] H. C. Cameron, *Guy's Hospital Gazette*, Dec. 22, 1923.

Ophthalmia Neonatorum

Etiology.—The commonest cause of ophthalmia is the gonococcus, the disease being contracted as the child passes through the infected maternal passages. Occasionally other organisms, such as the pneumococcus, streptococcus or B. coli, are the cause.

Clinical picture.—On the second or third day the eyes become red, swollen and begin discharging. A short acute course is run, and in three or four weeks the infection has settled, leaving a greater or less amount of damage behind, depending on the treatment. Ulceration of the cornea is the commonest complication, with perforation and then destruction.

All ophthalmic discharges during the first 21 days of an infant's life are notifiable, and 70 per cent. of such discharges have been found to be due to the gonococcus.

Treatment.—In the Metropolitan area there is a special hospital for ophthalmia neonatorum—St. Margaret's, Kentish Town, where the mothers of the patients are admitted and breast-feeding is continued. The routine treatment adopted is irrigation of the eyes with a solution of Eusol (1 in 7) at intervals of one, two or three hours, according to the extent of the discharge. After each irrigation one drop of a solution of acriflavine in castor oil (1 in 1,500) is instilled.

Prophylaxis.—A weak silver nitrate solution (say 1 per cent.), or a 5-per-cent. solution of Argyrol, instilled into the eyes at birth should act as an efficient prophylactic and prevent the disease.

Prognosis.—If taken early and properly treated the outlook for such cases is excellent.

Pemphigus Neonatorum (Bullous Impetigo)

Etiology and pathology.—Staphylococcal infections of the skin occur most frequently in early infancy, and may often be contracted from the infected finger of the maternity nurse or a cracked nipple in the mother. The infection spreads rapidly over the infant's body,

forming large bullæ containing thin pus (Fig. 4). Delay in treating these may prove fatal. Epidemics occur in institutions and may often be traced to one common source.

Treatment.—At the first signs of infection the infant should be removed from contact with other infants and the source of the infection sought. The blebs should be clipped if of any size, allowing the pus to drain, and the whole carefully powdered and kept as dry as possible. If the infection is extensive it will

Fig. 4.—Case of pemphigus neonatorum.

be found best to place the child in a warm bath of some weak antiseptic, such as Condy's fluid, several times daily, drying and powdering afterwards. Care should be taken to continue nourishing the infant to the utmost. A mixture of zinc oxide and starch powder, equal parts, will be found most useful. Boracic powder should be avoided as being hygroscopic and tending to keep the child moist.

Spastic Paralysis (Little's Disease)

The clinical picture of spastic diplegia is well known. It is seldom recognizable before the age of 6 or 8 months, when the child fails to sit up, but becomes increasingly evident from then onwards.

There are two definite groups, and the proportion of cases falling into one or the other cannot yet be ascertained.

(*a*) **Congenital cerebral aplasia.**—The first and better-known group consists of cases of *congenital cerebral aplasia.*[1] In such cases there is as a rule a greater or less degree of microcephaly and the brain shows marked under-development or abnormalities of the convolutions. These children have marked spasticity of all the limbs, the lower limbs being stiff and actually crossed in scissors fashion when the child is held up as if to walk. They cannot sit up and they make wild, uncontrollable movements with the arms and legs on any attempt to get them to do so. Their mentality may be slightly backward, or it may be that of an idiot or imbecile. They swallow badly, especially solids, and on this account they usually show signs of rickets. They are in many respects like the group to be described in the next paragraph.

(*b*) **Birth injury.**—These cases present a similar clinical picture to the first group, but are often much less marked or severe, and are due to *cerebral hæmorrhage at birth* (birth injury). Cerebral hæmorrhage at birth is probably much more common than is at present realized. Holland found hæmorrhage in 51 per cent. of infants dying as the result of difficult labour, and in half of these the tentorium cerebelli was torn.[2] Kennedy and Cruickshank found hæmorrhage in approximately 80 per cent. of 200 mature infants, gross in amount in 50 per cent.[3]

Etiology. *Trauma.*—While the head is passing through the maternal pelvis, great pressure is exerted on it from all sides, so that the skull-bones must overlap. The stress is borne not only by the skull-bones, but also by those partitions separating the various portions of the brain, such as the tentorium cerebri and cerebelli.

[1] James S. Collier, "The Pathogenesis of Cerebral Diplegia," *Proc. Roy. Soc. Med.* (Neur. Sect.), Oct. 11, 1923.

[2] Eardley Holland, Ministry of Health's Reports, 1922, No. 7.

[3] J. M. Cruickshank, *Lancet*, 1923, i. 836.

These partitions carry some of the great cerebral vessels, and if torn across, the vessels are torn also, allowing the blood to gush out. The brain is often ploughed up in this way.

Asphyxia.—If the labour is prolonged, and especially if the cord is about the infant's neck, the head becomes extremely congested. Bleeding takes place in these circumstances from even the smallest torn vessel.

Prematurity and delay in clotting of the blood.—In the premature infant the blood-vessels are fragile and poorly formed, and there is often a deficiency in the power of the blood to clot and thus to check the bleeding.

It has been shown that these factors are the most important in the production of cerebral hæmorrhage. After breech birth the incidence is high, but instrumental delivery does not appear to raise the incidence to any considerable extent.

Pathology.—The hæmorrhage may be extremely small, and present in some area of the brain giving rise to no symptom. On the other hand, the hæmorrhage may be so large that the brain is irreparably damaged. A clot may lie over the vertex of the brain, involving the motor areas, which accounts for the picture described as spastic diplegia. The tentorium or falx may be torn without any bleeding, merely causing the head to be misshapen.

Symptoms and diagnosis.—What symptoms would lead us to diagnose cerebral hæmorrhage immediately after birth? Inability to suck well, cry well, breathe properly, and a tendency to convulsions, are all most suggestive. Very often convulsions are postponed as late as the third or fourth day. The fontanelle tends to bulge, showing increased pressure within the brain. The mother probably first suspects that something is wrong when the infant is six to nine months old and makes no attempt to sit up. At a year, instead of taking more solid food, fluids only are taken. This is probably largely due to inco-ordination of the muscles of mastication and deglutition. The result is that the teeth decay easily and the bones tend to be soft. At

the age of two the child still does not sit up and may dribble from the mouth, making grimaces which suggest that he is mentally defective. It is usually not the case, however, as in the vast majority of these children the mentality proves to be normal or nearly normal.

Motor control.—If the child is picked up under the arms and held in the position as if about to walk, he assumes the "scissor-leg" attitude (Fig. 5). The adductors are in spasm, the toes are pointed, so that he rests his weight on his tip-toes. In time the tendo Achillis becomes shortened, the arms are stiff and all the movements are awkward. The grip is poor

Fig. 5.—Spastic diplegia (Little's disease), showing the "scissor-leg" position. (*By courtesy of Dr. Hector Cameron.*)

and the co-ordination of the muscles in making any fine movement is absent. The facial muscles and tongue are involved, so that speech is often impossible or poorly performed. Attempts at smiling or laughing tend to produce grimaces. When the child is asleep, however, all the muscles are flaccid, relaxed and normal. At the age of three or four the child has taught himself to sit up and gain balance. Solids are taken, he becomes shy of strangers, nervous and emotionally unstable. Walking takes place next.

Prognosis.—In children with very severe convulsions great damage has probably been done to the brain and they do not survive. Those who do survive may either recover completely or show a varying degree of spastic paralysis, as has been described. Those with Little's disease or spastic diplegia may live for a full lifetime, but if they are not properly cared for and carefully fed, death may occur from intercurrent diseases. Those able to get about may be normal or nearly normal mentally, and quite capable of earning their own living and becoming useful members of society. Complications such as convulsions, from which these patients are very prone to suffer, alter the prognosis for the worse.

Treatment.—In a normal-sized, full-term child, where the fontanelle is bulging, lumbar puncture is indicated. A valuable aid to diagnosis is an examination of the fluid withdrawn, noting its pressure. The fundus should also be inspected by means of the ophthalmoscope.

In a premature infant, if bleeding is continuous, the giving of 10 to 20 c.c. of the parent's blood beneath the abdominal wall may be indicated. In cases where operative treatment has been attempted in this country no good results have been reported. If convulsions are present chloral should be given rectally, 2 gr. every three or four hours until the child is drowsy. (For dosage by the mouth *see* Appendix, p. 488.) The problem is largely one of nursing, as the child must be fed and cared for until the bleeding has ceased and the clot has had time to be absorbed. This may take

several days. Feeding by stomach-tube is indicated where the infant makes no attempt to swallow. In most cases, however, it will take its feeds from a pipette or feeder. A sufficient quantity of food must be given to maintain life, and many patients at this stage die of insufficient food. No case should be regarded as so hopelessly damaged that an attempt to preserve life is useless. Often with continued and repeated convulsions for many days after birth a complete recovery will appear to be possible, and on the other hand, with symptoms which would suggest slight hæmorrhage only, much permanent damage has resulted.[1]

In older infants and children the upbringing is all-important. Much more patience is required than with the average infant, for in such matters as sitting up and feeding itself it is much behindhand. The mothering and education of the child are the things to which we must look for the greatest improvement. Where there are contractures, massage and manipulation are useful. Appropriate tenotomies should be performed when walking commences. The advice of an orthopædic surgeon should be sought at this time.

Cephalhæmatoma

Etiology.—This condition is caused by an extravasation of blood beneath the periosteum, due to pressure on the skull and scalp at birth. Asphyxia and congestion aid the formation of this hæmatoma.

Clinical picture.—As a rule the hæmatoma appears over the area presenting itself first during birth; occasionally it appears over that portion pressed on by forceps during delivery. This caput succedaneum is chiefly of interest because of its persistence, in some cases for as much as two to three months. It is most common in the occipital region, where, as it is gradually absorbed, a soft bulgy mass may be felt, the sides of which are hard and irregular and resemble a depressed fracture

[1] Donald Paterson, "Paralysis in Children due to Hæmorrhage into the Brain at Birth," *Journ. Soc. Massage and Medical Gymnastics*, Sept. 1926.

of the skull in the centre (Fig. 6). The contents of the mass are quite fluid and can be aspirated with a syringe and needle.

Treatment.—A cephalhæmatoma is best left untouched, a firm pad being applied in the early stages, and later no dressing is needed. Aspiration involves

Fig. 6.—Baby 5 weeks old with depressed fracture of the skull, afterwards successfully elevated.

the risk of infection and is best avoided. No permanent damage results and no further treatment is required.

Hæmatoma of the Sternomastoid

Etiology.—This hæmatoma is probably due to the rupture of some of the muscle-fibres and vessels during birth, the sternomastoid being slightly over-stretched.

Clinical picture.—A few days after birth a lump may be felt at about the middle third of the sternomastoid, or it may be noted that the child tends to keep the head tilted to one side, as if a torticollis were present, attention being drawn in this way to the extravasation of blood into the substance of the sternomastoid.

Prognosis and treatment.—Complete recovery occurs as a rule, but occasionally slight torticollis may

Fig. 7.—Erb's paralysis in an infant, showing the hand held in the "waiting for a tip" position. (*By the courtesy of Dr. Hector Cameron.*)

remain. The child should be placed to lie on the affected side, so that counter-stretching may result. No further treatment is necessary.

Erb's Paralysis

During the process of birth the child may be so manipulated that great stress is put on the brachial plexus by the head being flexed on the opposite shoulder, or the arm being unduly pulled upon. Either a hæmorrhage may occur into the plexus, causing temporary paralysis, or the plexus itself may be completely torn across, causing more or less permanent paralysis of that arm. In such cases, shortly after birth it is noticed that the arm is flaccid and held in a pronounced internally rotated position, with the wrist flexed. The arm thus assumes the position of a waiter's hand bent to receive a surreptitious tip (Fig. 7).

A Fairbank splint should be put on the arm and kept there for some months, electrical reactions being done to ascertain whether the nerve supply is returning to the affected group of muscles. If the muscles remain paralysed, some open operation must be performed on the plexus, the torn ends being joined together to prevent permanent paralysis. On the whole, if taken early, the prognosis is excellent.

Convulsions in the New-born

The commonest causes of convulsions in the newborn are cerebral hæmorrhages (*see* p. 41) and sepsis neonatorum (*see* p. 36), the latter giving rise to meningitis. Other causes are congenital brain malformations and nævi of the cerebrum. True epilepsy in a small proportion of cases may show itself first shortly after birth. It is not until three or four months at least have elapsed that convulsions due to tetany or spasmophilia play a part of any importance. Cerebrospinal and influenzal meningitis may both be found in the first few weeks of life, but tuberculous meningitis is rare before six months at least. Cerebral tumours are extremely rare under the age of one year. Cerebral degeneration, such as amaurotic family idiocy (Tay-Sachs' disease) is also rare, and seldom shows spasticity or convulsions until the child is about a year old.

Melæna Neonatorum (Hæmorrhagic Disease of the New-born)

This is not a common condition. It commences in the infant within the first week. Bleeding from the bowel is much the most common form, but it may be accompanied by the vomiting of blood. There may, however, be bleeding from the nose, or the umbilicus, or sudden effusion of blood into the suprarenal or some serous cavity. A rare occurrence is vaginal hæmorrhage occurring in the new-born. This does not accompany melæna neonatorum, nor does it fall into the same category. It must be looked upon, along with the early formation of milk in the breasts of the new-born, as a condition the cause of which remains unknown.

Etiology.—The cause is not known, but is supposed by some to be a form of infection, entering possibly through the umbilicus or by the bowel.

Pathology.—In cases with bleeding from the bowel and stomach, minute ulcers can be found at post-mortem. Many cases of sudden death have shown much hæmorrhage into the suprarenal.

Treatment.—This must be undertaken very promptly or the opportunity will be missed. The injection of 10 to 20 c.c. of whole blood beneath the abdominal wall is efficacious if done very early. If the condition has been allowed to go on the infant exhibits marked collapse and the transfusion should be carried out intravenously. A solution of 5 to 10 per cent. gelatin given by the mouth is said to be most effective. The author believes that the simplest method of treatment is to draw a 10-c.c.-syringe full of the whole blood from the mother or father and inject its contents quickly, before clotting can occur, beneath the subcutaneous tissue of the abdominal wall. With this treatment the mortality is extremely low.

Congenital Malformations

The frequency of congenital abnormalities among the general population is not accurately known, but

careful post-mortem examination shows the presence of these abnormalities in about 15 per cent. of the infants dying at the Hospital for Sick Children, Great Ormond Street, under the age of one year.[1] Of those dying under the age of 4 weeks (neo-natal cases), about one death in three is due to some congenital malformation.

In some cases there is malformation of a vital organ, such as the heart, kidney or bowel, which is incompatible with life. In other cases the abnormality, although gross, may not involve such important organs, the child's health being unimpaired.

Etiology and pathology.—Nothing definite is known as to the cause of congenital malformations. Recently Still has pointed out their frequency in first-born children.[2] In a review of some 50 mongols born immediately after the War, in the majority of cases the father had suffered from shell-shock or neurasthenia and the mother was young and healthy.[3] In pre-War cases the mother was elderly and at the end of her child-bearing period. An important contributing factor may be that one or other parent is sexually below normal. The development of the embryo or fœtus is thought to be interfered with mechanically or by toxins.

Variations of abnormality.—Among the more common congenital malformations are the various types of hernia, hare-lip and cleft palate, club-foot, congenital dislocation of the hip, nævi, congenital heart disease, congenital pyloric stenosis and congenital obliteration of the bile-ducts.

Treatment.—In some cases, such as hare-lip and cleft palate, the treatment is surgical; in others, such as heart disease and brain defect, nothing can be done; in others again, such as congenital pyloric stenosis or congenital laryngeal stridor, the congenital abnormality

[1] Donald Paterson, "Neo-natal and Post-natal Mortality," *National Health*, Vol. 16, No. 167, Aug. 1923, pp. 36-39.

[2] Still, *Lancet*, Vol. 213, Oct. 15, 1927, p. 795.

[3] Dora M. Berry, "An Investigation of 50 cases of Mongolian Imbecility from the O.P.D., Hospital for Sick Children, Great Ormond Street," *Brit. Journ. Child. Dis.*, Vol. 21, 1924, p. 259.

may right itself as the child grows older, although no specific treatment has been applied.

Congenital Absence of the Bile-ducts

This is a rare condition. Shortly after birth the child is seen to be slightly jaundiced and at first the case may be considered to be one of icterus neonatorum only. Then it is noticed that the jaundice is becoming more and more marked, the stools continue light in colour, and the urine is deeply stained with bile. The sclerotics have an icteric tinge. The spleen is, as a rule, palpable, and the liver, which is down several fingers' breadth below the costal margin, is hard and resistant on palpation.

Prognosis.—Despite the most careful feeding these children die at about the sixth or seventh month.

Pathology.—At post-mortem, in addition to a widespread distribution of bile, the liver shows a fine cirrhosis. It is of an olive-green colour, and on section under the microscope shows bile contained in the bile canaliculi. The gall-bladder may or may not contain bile, but if so it is small in amount. The common bile-duct may be actually patent, but as a rule it is represented by a fibrous cord.

There are cases, however, where preceding the jaundice there has been umbilical sepsis, and there is reason to think that the sepsis has passed to the liver, causing an obvious hepatitis with consequent cirrhosis. The clinical picture is very similar to that already described. Some observers consider that such cases are always the result of hepatic infection.

Treatment.—Beyond a diet extremely low in fat, which in some cases may prolong life, nothing appears to be of benefit. There are cases on record in which the gall-bladder has been joined up with the duodenum with good results. A laparotomy is generally resorted to in such cases on the assumption that possibly the stenosis of the common bile-duct can be relieved. It is seldom, however, that surgical interference is justified.

Congenital Atelectasis of the Lung

The normal condition of the lung before birth is one of collapse. At birth, with inspiration the lung becomes aerated and the process of expansion takes place rapidly within a few minutes, especially with vigorous crying. Failure of the lung to expand (atelectasis) is an uncommon occurrence.

Etiology and pathology.—The condition is most commonly due to some congenital malformation of the bronchi, a lobe or portion of a lobe remaining cut off from the bronchial tree. Such an area may present a cystic appearance at post-mortem, and may be free from air. Another cause is blocking of the bronchus leading to that portion of the lung by inspissated mucus or some foreign body. If the infant is feeble or premature it may have insufficient strength to aerate the whole of its lung tissue fully.

Clinical picture.—An infant with congenital atelectasis may present few or no symptoms, but if it be premature or weakly its other symptoms may be aggravated by the pulmonary condition. Sudden "blue turns" or attacks of dyspnœa, such as are seen in congenital heart disease, may occur. An infant with extensive atelectasis, but otherwise perfectly normal, sooner or later presents a picture of respiratory insufficiency, dyspnœa on slight exertion, a tendency to cyanosis and a cough, with bronchitis to a greater or less degree. On examination there is dullness on percussion over the affected part and displacement of the mediastinal contents towards that place. On auscultation the breath-sounds are tubular, being conducted thus by the solid substance of the lung. X-ray examination confirms the picture. In older children it is difficult to differentiate between the appearances described and those due to pulmonary fibrosis following on unresolved pneumonia. In a new-born infant, however, a pulmonary fibrosis is uncommon.

Prognosis.—If the atelectasis is only of a small

portion of a lobe it is not incompatible with life and may even escape detection for many years. If a lobe or more is involved, however, the symptoms are sufficient not only to attract attention but to cause serious ill-health. A severe degree of atelectasis in a new-born infant often leads to death.

Treatment.—Where there is some congenital malformation nothing can be done, but if mucus is present it may be loosened and its removal aided by the inhalation of steam or the giving of small doses of an expectorant. Inversion of the child helps to drain away obstructing mucus. The mode of life of such children is like that of the child with congenital heart disease, and symptoms must be treated as they arise.

THE CARE AND FEEDING OF THE PREMATURE INFANT[1]

To have a clear idea of the difficulties of the care and feeding of the premature infant it is necessary to consider, first, why the child has been born prematurely, and the possible effects on the child of these pathological factors, and secondly, what damage may have occurred to the infant during or after birth. Some of the factors which, acting through the mother, may produce premature labour, are syphilis, nephritis (toxæmia of pregnancy), heart disease, acute infectious fevers, chronic infections of the uterus, multiple pregnancy (twins or triplets), poisoning of the mother by lead, arsenic or other metals. Malformations or abnormalities in the fœtus itself, such as hydrocephalus, or absence of the abdominal wall, may cause premature birth. Miscarriage occurs sometimes without any known cause. To realize what damage may occur to the infant at birth one must consider the possibilities of hæmorrhage, especially cerebral hæmorrhage, occurring during labour. In the premature infant the vessels are so poorly formed and

[1] The substance of this section has appeared in the *Post-Graduate Medical Journal*, June 1927.

supported that, although labour may be extremely easy, trauma and pressure are withstood badly and the slight moulding of the head during birth tends to pull on and tear the fine vessels of the brain and meninges, causing hæmorrhage. Another factor is the tendency for premature infants to bleed. The coagulation time is lengthened in many premature infants, and also the "bleeding time." Whether this is due to the fact that these infants suffer from a deficiency of calcium, since that salt is largely given by the mother in the last two months of intra-uterine life, it is difficult to say. Certainly the incidence of hæmorrhage at birth is much higher in premature than in full-term infants. Very often the hæmorrhage gives rise to a clinical picture resembling spastic diplegia, or the infant develops hydrocephalus with spastic paralysis down one side of the body.

Care of the premature infant.—In the management of the infant the chief factors are the preservation of its body-heat, that is, the prevention of a subnormal temperature, and careful administration of sufficient and suitable food.

Methods for the maintenance of body-temperature.—The premature infant has no means within itself of keeping its temperature up to normal. Heat must be applied then from outside. Its breathing is so shallow, its circulatory system so inactive and its metabolism so slowed that it is not able to manufacture heat for itself. One of the chief problems therefore with these infants is to keep their temperature within normal limits.

The baby should be received at once into a jacket of cotton-wool, both arms and legs being done up in this material, and the buttocks being kept free with a separate pad to be used as a napkin. The infant is then wrapped round in a shawl and the whole deposited in a basket or bassinette, for which some method of heating has been devised. A simple method for poor parents is to hang several stone ginger-beer bottles, filled with hot water, just within the basket. Two

or more hot-water bottles placed one at each side and one at the foot of the basket and changed frequently should suffice to keep the baby's temperature up to normal. A powerful electric light suspended over the infant, which has been covered by a blanket or tarpaulin, is another method advocated by some. The author has found the simpler means the better.

No attempt should be made to bath a premature infant during the first few weeks of life. It should be kept well rubbed with olive oil and the buttocks sponged from time to time, the whole child well wrapped in its cotton-wool covering. Weighing should be done in its clothing, and when an opportunity arises and the clothing is changed the weight of this can be deducted, so that the weight of the actual child is then ascertained. When the infant has attained the weight of 5 to 6 lb. normal clothing can be used.

The question whether premature babies should be admitted to hospital or not is a most debatable one. Is there any great virtue in the institutional treatment of such cases? Can anything more be done in hospital than in a private house where an intelligent nurse is in charge? It is doubtful. Cases treated in incubators and superheated rooms specially prepared for the purpose have not in the author's experience had greater success than those treated in a private house and properly cared for by an intelligent mother, however poor she may be.

The feeding of the premature infant.—The caloric requirements of the premature infant are vastly higher in proportion to its body-weight than those of a normal infant. The problem is reduced therefore to one of securing for the infant a food sufficiently high in caloric value to make it gain, and sufficiently digestible to allow the intestinal tract to cope with it in large enough quantities to promote growth. The line between overfeeding and keeping the intestinal tract healthy, and underfeeding but supplying sufficient calories, is a very fine one. The slightest swing to either side proves equally disastrous. It is difficult to say whether more premature infants

are lost from underfeeding or from overfeeding—probably from the former, as it is not commonly known that such infants require relatively enormous quantities to thrive. From the above remarks it is apparent that the great digestibility of breast-milk singles it out as ideal for the feeding of these babies, since it can be dealt with in relatively large quantities, without fear of digestive upset, in a degree with which no other food can compare.

Breast-feeding.—There is no doubt that where the mother can breast-feed her infant the chances of its survival are greatly increased. Very often, however, if the infant be very premature, in which case suckling is all the more necessary, the mother is quite unable to supply breast-milk, as it has not appeared. This is especially true in the case of the first baby. In such circumstances a wet nurse should be sought. On the Continent this is not a very difficult problem, but in England the greatest difficulty is often experienced in procuring a wet-nurse. Some of our institutions have paid wet-nurses, and there is no other reason for advocating institutional treatment for a premature baby than the possibility that breast-milk can be supplied. Friends or neighbours will occasionally part with some of their breast-milk for the time being, however, and give the infant a start. Should the mother be able to breast-feed the child or should a wet-nurse be obtained, it should be put to the breast twice in the second 12 hours, and thereafter at three-hourly intervals for a gradually lengthening period, commencing with three minutes. If the infant is too feeble and undersized to suck properly, the breast-milk may be expressed and fed as described under Artificial Feeding. It is well known that a strong healthy infant sucking at one breast tends to make the milk flow from the opposite breast. This fact may be taken advantage of, and a wet-nurse, by placing her strong infant to one breast and at the same time placing the premature infant to the other breast, may cause the milk to flow into the mouth of the latter with little or no effort.

Times of feeding.—Certainly for the first fortnight the premature infant on the breast will require to be fed as follows: if under 3½ lb., two-hourly feeding by day and three-hourly feeding by night, and if over 3½ lb. three-hourly feeding by day and four-hourly feeding by night. More frequent feeds are required where artificial feeding has to be carried out.

Amount of breast-milk required.—A normal healthy infant requires about 2½ oz. per pound of its body-weight per day, that is, an infant weighing 6 lb. at birth very quickly obtains about 15 oz. of breast-milk in the day from its mother ($2\frac{1}{2} \times 6$). The premature infant requires relatively a greater amount in the day for each of its pounds and fails to thrive on anything less than about 3 oz. per pound body-weight per day; that is, a premature baby weighing 4 lb. will require about 12 oz. of breast-milk in the day before it begins to gain weight properly. Waiting for the milk to appear, i.e. until the third or fourth day, is more trying for the premature infant than for the normal infant. Complementary feeds, therefore, or the giving of additional feeds after the breast, should be commenced after 24 hours until the mother's breast-milk can take their place. In the second 24 hours the infant requires 1½ oz. for each of its pounds in the day, whereas by the fourth 24 hours this has risen to 2 oz. per pound body-weight per day. By a fortnight at latest the full amount of 3 oz. per pound body-weight per day should have been reached.

Artificial feeding. *Choice of food.* — The premature baby has not the same ability to manage the various food elements as the full-term child. Sugar is metabolized efficiently, and relatively high carbohydrate feeds are to be recommended. Protein or curd, however, must be very carefully modified, as there is no doubt that the infant is most susceptible to curd indigestion in the first few weeks of life. Fat seems to be managed in a moderate manner. Probably the most useful food for the premature infant is unsweetened condensed milk. "Ideal" or Libby's milk 1 oz., water

3 oz., sugar one rounded teaspoonful, make an almost humanized mixture, and the child should gradually be given an amount equal to 3 oz. of this for each of its pounds in the day. This amount should be reached by about the 14th day. An example would be that of a 4 lb. premature baby requiring 12 oz. of the mixture in the day. The child is to receive 10 feeds, and therefore, starting with one ounce at each feed, the amount can be increased by adding an extra teaspoonful to each feed until the whole 12 oz. are given daily. The daily weighing of the child is a most necessary precaution. Should the infant show a steady gain no increase in the feed should be given, but if the weight is stationary, then the feed should be cautiously increased. Other foods also successfully used are the humanized dried milks. These are made up so that one very heaped teaspoonful dissolved in 1 oz. of water reconstitutes as near as possible 1 oz. of breast-milk. Some of the better known of these foods are Allenburys' No. 1, New Glaxo, Recolac, Humanized Trufood, Almata and Half Cream Cow and Gate. It is obvious that the amount required of these would be 3 heaped teaspoonfuls dissolved in 3 oz. of water per pound body-weight per day.

A useful food, but less well known, is lactic acid milk. This is made as follows: To one pint of boiled skimmed cow's milk, which has been allowed to cool, 45 drops of B.P. lactic acid are added, drop by drop, stirring continuously. This mixture is diluted with an equal quantity of water, making it up to a quart, and a suitable quantity of sugar is added, as a rule one heaped teaspoonful to each 4 oz. of the mixture. As the child becomes accustomed to this the strength ought to be increased to two parts of the milk to one of water in a few weeks' time. The lactic acid milk will be found extremely useful in the feeding of premature infants, especially when there has been some slight diarrhœa or vomiting. Fully peptonized cow's milk or full-cream dried milks to which lactic acid has been added are also useful, but these require great care in preparation and are probably unduly complicated.

Times of feeding.—The infant under 3½ lb. should be fed at 6, 8, 10, 12, 2, 4, 6, 9, 12, and 3. When thriving well these periods should gradually be lengthened until the infant is fed three-hourly by day and four-hourly by night.

To give an example of the feeding of a particular infant let us take a case such as this. An infant is born one month prematurely, weighing 3 lb. This infant should be fed at 6, 8, 10, 12, 2, 4, 6, 9, 12 and 3, and be given ultimately 9 oz. of breast-milk in the day, or 9 oz. of the "Ideal" milk mixture, or 9 oz. of lactic acid milk. Supposing the "Ideal" milk mixture is the most suitable, commence by giving half an ounce in a bottle with an easy hole in the teat, at each feed. Then the child should be weighed each day, which of course necessitates a pair of scales in the home. A spring balance is quite good enough. Each day the bottles should be increased by one teaspoonful; that is, having got the child accustomed to half an ounce by the fourth or fifth day, 5 drachms should be given and, if the child is not gaining, increased to 6, then to 7, and finally to 8 drachms. As soon as the infant commences to put on weight, an ounce per day or more, no further increase in the feed will be made, but at any sign of stationary weight the feed should be once more increased by a further drachm.

Difficulties of administering the feeds.—In some infants there is obstinate anorexia. The baby sleeps continuously and there is no desire for food. The mouth should be examined carefully, to make sure it is not sore. It may be necessary for a period of 24 hours to give half-normal saline or boiled water sweetened with sugar. Should the infant be extremely weakly and unable to suck the breast or bottle properly, the Breck Feeder, or what is known as the Premature Tube Feeder, sold by Messrs. John Bell and Croyden, will be found a great help. This is made by placing on the end of a glass tube a small teat made from the rubber end of a fountain-pen filler, in which a very fine hole has been pierced. On the other end of the glass

tube an unperforated teat or finger-stall is placed and the tube filled with milk. Gentle pressure on the unperforated end forces the milk to flow from the opposite end of the tube, and the baby gets its food with little or no work. Glaxo and other firms place on the market a bottle for premature babies which is supplied with extremely small teats and is most useful. Should the infant refuse all fluid for any length of time it may be given its food by nasal or gastric tube. This is very seldom necessary.

"Blue turns" in premature infants.—Premature infants are extremely liable to sudden attacks of cyanosis, or "blue turns." There may be a variety of causes for this. The infant may have had a slight hæmorrhage round about the respiratory centre in the medulla. Or the trouble may be due to a congenital atelectasis of the lungs, a portion of one or both having failed to expand. The child may have congenital heart disease, that is, owing to its prematurity there may be a communication between the two sides of the heart or a patent ductus arteriosus. Finally, attacks of sudden cyanosis occur in infants if they become unduly dehydrated. This is seen occasionally where an infant has refused its food or has slept so soundly that it could not be wakened for its feeds. Insufficient fluid is taken, and on possibly the second, third or fourth day a sudden attack of cyanosis with difficulty in breathing occurs. The giving of fluids to such a case will rapidly right it. Various observers, however, have quoted cases in which there have been blue turns proving fatal and no explanation can be offered[1]. When these attacks of cyanosis come on the infant is best kept quiet, fed in its cot and not picked up or handled in any way. Oxygen should be administered, and in cases of extreme urgency it may be necessary to give a mustard bath and resort to artificial respiration.

Prevention of rickets in premature infants.—In the last two months of intra-uterine life the infant

[1] G. F. Still, "Attacks of Arrested Respiration in the New-born," *Lancet*, 1923, i. p. 431.

receives both calcium and iron in large quantities from its mother. Should it be born prematurely it is of necessity short of calcium and iron and therefore both anæmic and an incipient case of rickets. The incidence of fully-developed rickets in premature infants is extremely high. This need not be. From birth the infant requires small doses of cod-liver oil; a half-teaspoonful of a well-made emulsion twice daily will suffice. Where possible, artificial sunshine should be given, and in summer-time full advantage should be taken of direct sunshine. From an early age raw meat juice or bone and vegetable broth should be added to the midday bottle. This can be done safely after one month, in teaspoonful doses, gradually increased until two tablespoonfuls are being given. Strong milk mixtures are preferable to very weak ones, and if the child be breast-fed, additions of cereals and soups to the mother's breast-milk should be made at an early date.

CHAPTER III

THE FEEDING OF THE NORMAL INFANT AND CHILD[1]

It should be taken as a general principle that every infant should be breast-fed, wholly or at least in part. Where, for some reason, this cannot be carried out, certain principles of artificial feeding must be considered. The importance of understanding the fundamentals of infant feeding is being more and more appreciated, both by the doctor and the general public. No practitioner can be said to be a properly trained doctor without a full knowledge of these principles.

TABLE VI

COMPOSITION OF MILK FROM DIFFERENT MAMMALS

(Modified from "Abt's Pediatrics")

Mammal	Specific Gravity	Water	Casein	Albumin	Total Protein	Fat	Sugar	Ash	Total Solids
Man	1.0298	87.58	0.80	1.21	2.01	3.74	6.37	0.30	12.42
Cow	1.0313	87.27	2.88	0.51	3.39	3.68	4.94	0.72	12.73
Ass	1.032	90.12	0.79	1.06	1.85	1.37	6.19	0.47	9.88
Goat	1.0305	86.88	2.37	0.89	3.76	4.07	4.64	0.85	13.12
Mare	1.0347	90.58	1.30	0.75	2.05	1.14	5.87	0.36	9.42

Cow's Milk

Graded milks.—1. Certified milk is produced from cows which have passed the tuberculin test and

[1] Most of the tables in this chapter have already appeared in Paterson and Forest Smith's "Modern Methods of Feeding in Infancy and Childhood" (Constable & Co. Ltd.), where the whole subject of infant and child feeding is dealt with in much greater detail.

a veterinary examination. These cows are tested and examined at intervals of six months. The milk is bottled on the farm and there must not be more than 30,000 organisms per c.c.

2. Grade A milk (tuberculin tested), produced under the same conditions as certified milk, must not contain more than 200,000 organisms per c.c.

3. Grade A milk is similar to No. 2, but the tuberculin test is not required. A veterinary examination of the herd takes place every three months.

Is it necessary to boil cow's milk?—By boiling cow's milk the curd is made more digestible. It is true that the vitamins are slightly diminished in the process, though not destroyed, but these are easily made good by giving fresh fruit juice and a little cod-liver oil, the latter especially during the winter months. The chief advantage of bringing milk to the boil is that all pathogenic organisms are thus killed off. The commonest and most to be feared is the tubercle bacillus, but epidemics of diphtheria, scarlet fever and infantile paralysis have all been traced to the milk supply, and the simple precaution of scalding the milk prevents infection from this source. The author considers that milk given to every child up to the age of two should be boiled. After this age, if certified milk is used, boiling is optional.

Condensed milks.—These milks are manufactured by heating milk and evaporating it in vacuo. Once a tin of the unsweetened variety has been opened it will not keep longer than fresh cow's milk. The sweetened varieties, however, because of their high sugar content, will keep considerably longer.

The composition of a number of the better-known brands of condensed milk is given in Table VII.

Sweetened full-cream condensed milks.—In this group the vitamins have been almost entirely conserved, and the milk is sterile. The tendency in making up such milks, which have added sucrose, is for the protein and fat content to be low compared with the high carbohydrate content. A little fresh

cow's milk and cod-liver oil emulsion should be added to the diet of infants fed on these milks; they then constitute a satisfactory food for the normal infant.

Unsweetened full-cream condensed milks.—The milks in this group are extremely useful in feeding underweight or premature infants, as the curd is very digestible. Cod-liver oil and fresh fruit juice must be given with them, however, and it must be remembered that once the tin is opened the milk will not keep. Made up in the proportion of one part of condensed milk to three parts of water, with a heaped teaspoonful of sugar to each 4 oz. of the mixture, a humanized mixture is obtained.

Sweetened skimmed condensed milks.—Such condensed milks are unsuitable for feeding to the human infant without some additions, but if cow's milk and cod-liver oil emulsion are also given their deficiencies are made good. Fruit juice should always be given to infants so fed. Older children who are thin and under weight and have a poor tolerance for fat sometimes thrive on such milks, spread thickly on bread and butter in place of jam.

Dried milks.—During the process of drying cow's milk the curd is modified to some extent, so that it becomes more easily digested by the infant than fresh cow's milk. Dried milks are manufactured in two ways: either by pouring fresh milk on hot rollers or by the spray process, in which it is dried by forcing the milk through small jets into a hot atmosphere.

The advantages of dried milk are: (1) It is sterile and tends to remain so over long periods; (2) in the process of drying the curd has been modified and is more digestible than in fresh milk; (3) it is easy to keep a supply, and this is a great advantage when travelling or when fresh milk cannot be properly kept; (4) the vitamin loss is very small and in many brands there is no loss at all; moreover this old objection is rapidly disappearing, as cod-liver oil and fruit juice can be given as a safeguard.

The chief *disadvantage of dried milk* is the cost, which is always greater than that of fresh milk.

Full-cream dried milk.—For practical purposes these dried milks are made so that one part dissolved in eight parts of water reconstitutes whole cow's milk, that is, a measure or drachm dissolved in an ounce of water reconstitutes an ounce of cow's milk. The composition of some well-known brands of dried milk is given in Tables VIII, IX and X.

TABLE VII

COMPOSITION OF THE BETTER-KNOWN CONDENSED MILKS

Name of Milk	Calorie value of 1 ounce	Fat per cent.	Protein per cent.	Carbohydrate per cent.	Water per cent.
A. SWEETENED FULL-CREAM CONDENSED MILKS					
Nestlé's	102	10.2	9.4	53.0	25.3
Diploma	101	9.6	8.4	54.62	25.5
Berna	103	10.5	9.5	53.0	25.0
Tip-top or Red Butterfly	98	9.1	8.54	52.86	26.9
B. UNSWEETENED FULL-CREAM EVAPORATED MILKS					
Ideal	51	9.5	8.84	12.21	67.5
Carnation	48	9.2	8.6	11.1	69.4
Libby's	50	9.2	9.6	11.5	68.0
Regal or Coronet	50	9.6	8.32	12.02	68.2
Berna	49	9.5	9.5	10.0	69.0
Everyday or Green Butterfly	50	9.1	8.85	12.20	67.9
Pet Milk	42	8.0	7.6	9.7	73.4
C. SWEETENED SKIMMED CONDENSED MILKS					
Moonraker	84	0.55	9.76	59.84	27.6
Blue Butterfly	84	0.4	10.0	58.8	28.8

Humanized dried milks.—These are made so that one teaspoonful or measure dissolved in an ounce of water reconstitutes an ounce of breast-milk as nearly as possible.

Starchy patent foods.—The majority of the starchy patent foods are made up by adding them to cow's milk. Some require cooking, while others, such

TABLE VIII

COMPOSITION OF THE BETTER-KNOWN FULL-CREAM DRIED MILKS

Name of dried milk	Water per cent.	Protein per cent.	Fat per cent.	Carbo-hydrate per cent	Calorie value of 1 ounce
Glaxo (full-cream)	2.4	22.9	25.5	42.5	147
Lactogen	3.0	23.7	24.5	43.6	147
Trufood (full-cream)	1.97	24.02	26.0	41.9	149
Dorsella	2.2	22.3	27.1	42.3	151
Neave's Milfo	2.4	20.0	26.0	47.1	151
Cow and Gate (full-cream)	2.5	26.6	27.3	37.6	151
Lacta	3.6	26.25	26.05	37.0	146
Milkal	1.5	26.05	29.0	37.64	155
Ambrosia	1.6	28.7	29.3	35.2	156
Klim (full-cream)	1.5	26.7	28.2	37.9	154
Virol and Milk	2.7	26.5	28.0	37.5	156

TABLE IX

DRIED MILKS WITH A LOW FAT-CONTENT

Name of dried milk	Water per cent.	Protein per cent.	Fat per cent.	Carbo-hydrate per cent.	Calorie value of 1 ounce
Cow and Gate (skimmed)	3.3	35.2	0.8	52.8	108
Glaxo (half-cream)	2.0	31.5	16.5	43.5	135
Trufood (skimmed)	4.0	36.0	1.0	49.0	105
Trumilk (half-cream)	1.85	32.0	13.86	45.1	130
Dryco	3.0	34.0	12.0	44.0	126
Klim (skimmed)	2.8	37.7	1.4	49.9	109
Horlick's Malted Milk	2.5	13.9	8.03	71.59	124
Hooker's Malted Milk	1.5	14.0	9.2	81.8	140
Secway	1.2	14.25	0.27	72.26	104
Casec	4.12	89.0	1.6	—	111

TABLE X

THE COMMONER "HUMANIZED" DRIED MILKS AND THEIR COMPOSITION

Name of dried milk	Water per cent.	Protein per cent.	Fat per cent.	Carbohydrate per cent	Calorie value of 1 ounce
Cow and Gate Half Cream .	2.5	20.0	15.5	58.0	135
Allenburys' No. 1 . .	5.7	9.7	18.5	62.3	136
Allenburys' No. 2 . .	3.9	9.2	17.6	66.8	139
New Glaxo . . .	2.0	17.0	20.0	56.0	141
Humanized Trufood . .	1.4	11.8	28.9	52.3	155
S.M.A.	1.0	9.0	28.0	58.0	156
Humanized Ambrosia . .	1.3	14.4	25.6	55.2	153
Recolac	2.0	12.0	27.0	57.0	156
Berina Food No. 1 . .	3.04	19.74	17.30	55.95	137
Berina Food No. 2 . .	3.04	20.35	15.77	56.92	135
Almata	3.3	12.5	27.0	54.6	153

as Savory & Moore's Food, need only be added to the warm milk mixture and allowed to stand. Such patent foods are extremely useful to introduce starch into the diet of the infant after the sixth month, and they may also be used in the early months of life to thicken the food of babies addicted to rumination (*see* p. 140).

In the following summary the percentages of protein (P.), fat (F.), and carbohydrate (C.) only are mentioned, the remaining figures being supplied by the ash content and moisture. It must be remembered that the dried preparation only is represented by these analyses, and that the excess of sugar and starch is greatly modified when the food is made up by the addition of milk and water.

COMPOSITION OF PATENT FOODS

Allenburys' Malted Food, No. 3.—(P.), 9.2 ; (F.), 1.0 ; (C.), 82.8.—A mixture of wheat flour and malt. When prepared according to directions, it still contains some unaltered starch. Designed for children above the age of six months. One tablespoonful (about 1 oz.), a teaspoonful of sugar, and three tablespoonfuls of cold water ; mix, and add ½ pint of boiling milk and water (equal parts). Calorie value of 1 oz. = 113.

Arrowroot.—(P.), 8 ; (F.), trace ; (C.), 82.5.—Made from the starch of the root of a West Indian plant (Maranta arundinacea). Calorie value of 1 oz. = 100.

Benger's Food.—(P.), 10.2 ; (F.), 1.2 ; (C.), 79.5.—A mixture of wheat flour and pancreatic extract. When prepared according to directions, most, but not all, of the starch is converted into soluble forms. The protein is also partially digested as well as that of the milk used in mixing it. One tablespoonful (about 1 oz.) and four tablespoonfuls of cold milk, then add ½ pint of boiling milk and water ; set aside in a warm place for fifteen minutes, then bring to the boil. Calorie value of 1 oz. = 110.

Chapman's Whole Wheat Flour.—(P.), 9.4 ; (F.), 2.0 ; (C.), 79.3.—A finely-ground wholemeal flour. Not much superior in nutritive value to ordinary "household" flour. Starch entirely unaltered. Calorie value of 1 oz. = 112.

Cream of Rice (Groult's).—(P.), 6.9 ; (F.), 0.3 ; (C.), 80.0.—This is a patent food containing a high proportion of starch. Calorie value of 1 oz. = 105.

Cream of Wheat.—(P.), 11.81 ; (F.), 2.40 ; (C.), 72.46.—Made from the granulated endosperm, or kernel, of wheat. Calorie value of 1 oz. = 108.

Glaxo Malted Food.—(P.), 11.3 ; (F.), 2.1 ; (C.), 81.6.—A partly malted food prepared from wheat and barley. Calorie value of 1 oz. = 117.

Glax-Ovo.—(P.), 18.3 ; (F.), 13.3 ; (C.), 62.2.—A preparation consisting of milk solids, malt extract, flavoured with chocolate and containing added vitamin D. Calorie value of 1 oz. = 133.

Mellin's Food.—(P.), 10.3 ; (F.), 0.16 ; (C.), 79.5—A completely malted food. All the carbohydrate is in a soluble form. May be regarded as a desiccated malt extract. Half a tablespoonful, ¼ pint of milk and ¼ pint of water for a child under the age of three months. Calorie value of 1 oz. = 108.

Neave's Cereal Food.—(P.), 13.04 ; (F.), 0.97 ; (C.), 80.84.—A purely cereal preparation. Calorie value of 1 oz. = 115.

Nestlé's Milk Food.—(P.), 14.5 ; (F.), 6.15 ; (C.), 77.2.—A mixture of desiccated Swiss milk, baked wheat flour and cane sugar (27 per cent.). Contains about 18 per cent. of starch. Calorie value of 1 oz. = 126.

Ovaltine.—(P.), 14.2 ; (F.), 8.01 ; (C.), 67.9.—A concentration of the nutritive constituents of malt, milk and eggs, flavoured with cocoa, and issued in the form of light, readily soluble granules. Contains neither starch fibre nor husks of grain. Calorie value of 1 oz. = 120.

Ridge's Food.—(P.), 9.2 ; (F.), 1.0 ; (C.), 81.2.—A baked flour, containing only 3 per cent. of *soluble* carbohydrates, the remainder being starch. Recommended to be made with milk and water. Calorie value of 1 oz. = 111.

Robinson's Patent Barley.—(P.), 5.1 ; (F.), 0.9 ; (C.), 82.0.—Ground pearl barley, poor in every element except starch and mineral matter. Calorie value of 1 oz. = 106.

Robinson's Patent Groats.—(P.), 11.3 ; (F.), 1.6 ; (C.), 75.0.—Ground oats from which the husk has been removed. Rich in protein and mineral matter. Calorie value of 1 oz. = 110.

Ryvita Crispbread.—(P.), 11.6 ; (F.), 1.3 ; (C.), 74.8.—Made in Sweden from crushed whole rye grain. Calorie value of 1 oz. = 107.

Savory & Moore's Food.—(P.), 10.3 ; (F.), 1.4 ; (C.), 83.2.—Composed of wheat flour with the addition of malt and diastase. When prepared according to the directions, most, but not all, of the starch is converted into soluble forms (chiefly dextrins). One or two tablespoonfuls (equals from 1 to 2 oz.) to be mixed with two or three tablespoonfuls of cold milk or milk and water, and ½ pint of boiling milk or milk and water to be added. Calorie value of 1 oz. = 115.

Scott's Oat Flour.—(P.), 9.7 ; (F.), 5.0 ; (C.), 78.2.—A fine oat flour. Starch unaltered. Calorie value of 1 oz. = 118.

Sister Laura's Food.—(P.), 15.4 ; (F.), 1.98 ; (C.), 79.9.—A food prepared from wheat starch, intended as an addition to undiluted milk. Calorie value of 1 oz. = 120.

It should be pointed out that some infants have a low tolerance for carbohydrate in the form of sugar, but are able to tolerate it when given in the form of starch. These infants do well when starchy foods are added to their diet.

BREAST-FEEDING

The *preparation of the nipples* should commence two months before parturition. They should be scrubbed daily with a very soft boiled nail-brush and cold water frequently applied. In this manner they become erect and can be gently pulled upon if there is a tendency to retraction. A little Friar's balsam should be applied if there is any tendency to become sore.

Colostrum.—This is the fluid secreted by the breasts in the first few days after childbirth, and is high in protein and low in fat and sugar content. It is possible that it conveys immune bodies to the infant, but the exact significance of the colostrum is not understood.

TABLE XI (Holt)

SHOWING COMPOSITION OF COW'S AND HUMAN MILK AND VARIATIONS WHICH MAY OCCUR IN THE COMPOSITION OF BREAST-MILK

Components	*Cow's Milk* Per cent.	*Human milk* Per cent.	*Common healthy variations in human milk* Per cent.
Water	86—87	88.05	87.82—85.50
Fat	4.00	3.50	3.00— 5.00
Proteins . . .	3.50	1.25	1.00— 2.25
Milk sugar . . .	4.50	7.00	6.00— 7.00
Mineral salts . .	0.75	0.20	0.18— 0.25

Breast-milk.—It will be seen from Table XI that the fat-content of breast-milk varies from 3 per cent. to 5 per cent. The globules, however, are extremely small compared with cow's milk and the emulsion is much finer. This makes its digestion a much easier matter. The fat content of the milk cannot be varied by giving a richer or poorer diet to the mother, provided she is adequately fed. Protein is present to the extent of $1\frac{1}{2}$ to 2 per cent. compared with $3\frac{1}{2}$ to 4 per cent. in cow's milk. The bulk of the protein is present as lactalbumin and lactglobulin, and only a small proportion as casein. In cow's milk, on the other hand, the casein content is from four to five times as great as the albumin content. The clot formed in the stomach of the infant on breast-milk is extremely small. By diluting cow's milk and therefore reducing the protein from 4 to 2 per cent., an attempt may be made to approximate cow's milk to breast-milk, but clearly the proportion of insoluble casein in cow's milk cannot be made to approximate to that of breast-milk. Attempts at humanizing milk by dilution can never be strictly accurate. The carbohydrate present in breast-milk is lactose. The amount present in breast-milk is approxi-

mately 7 per cent., which gives breast-milk a much sweeter taste than cow's milk. The salts in breast-milk are present in a much smaller quantity than in cow's milk. Unfortunately, at the present time, our knowledge of the rôle played by these salts is extremely elementary.

Infants feeding at the breast seem to have a much higher *immunity* to disease than those fed on artificial food. It would appear possible, therefore, that some immunity to diseases can be passed on from the mother to her infant through the medium of the breast-milk, but of this it must be admitted there is no scientific proof.

Normal breast - feeding. — Breast - feeding suits nearly every infant, and most women are able either wholly or partially to breast-feed their children. When on account of social duties or of temperament and general make-up the mother is unable wholly to breast-feed her child—and in some cases the longer the attempt is continued the more the child deteriorates—*complementary feeding*, that is, giving cow's milk at the end of the breast-feed, may be adopted with great benefit. The individual mother must be studied, just as the individual child is studied in artificial feeding.

Contra-indications to breast-feeding.—Where the mother has open tuberculosis, as there is a risk of infecting the child, she should not attempt breast-feeding. A syphilitic infant should be nursed by its own mother. In acute infections, such as typhoid fever or pneumonia or puerperal septicæmia, the mother is too ill to breast-feed, but in mild infections there is no contra-indication. In heart disease, nephritis, anæmia and epilepsy, each individual case must be considered. The onset of menstruation during lactation should not be taken as an indication for weaning. The infant may require to be given artificial food for 24 hours, but should resume its breast-feeds thereafter. The intervention of pregnancy during lactation is not in itself a contra-indication, and should the mother and the infant both thrive, weaning is unnecessary. Should either, however, show signs of failing, weaning should take place at once.

Variation in quantity and quality of breast-milk (*see* Table XI, p. 70).—The first feed in the morning is probably the largest in quantity. From that time the feeds get smaller and in the middle of the day reach their smallest, remaining deficient until the evening, when the amount again increases. The quantity of the milk seems to vary with the activity of the mother, but the composition of the milk would appear to be constant for any particular woman.

Lactation and its establishment.—During the second 12 hours after the birth of the infant it should be put to the breast twice. *It is the sucking of the infant which stimulates the flow of milk, and without this strong suction the establishment of lactation is impossible.* After the first 24 hours regular times of feeding should commence, three-hourly or four-hourly as the case may be, and both breasts should be used at each feed.

Complementary feeds, and when they should be started.—Once the flow of milk is established, if it seems plentiful one breast alone can be used for each feed, the infant being kept there about 15 minutes. Very often, however, there is a delay in the establishment of the flow of milk, and during this time the child cries constantly, is inclined to have loose stools and to swallow much wind, with the result that breast-feeding is often abandoned. The weight of the child should be taken daily, and if the loss continues beyond the fourth day, small complementary feeds should be given after each breast-feed. This, however, should be merely a temporary measure, to be adopted only until the flow of milk is fully established, when it can be discontinued. Such a complementary feed might consist of a tablespoonful of boiled cow's milk, two tablespoonfuls of water, with a quarter of a teaspoonful of sugar.

Times of feeding.—The time and frequency of feeding must obviously depend on the size of the child and the supply of breast-milk. If the child is small, naturally its stomach holds an insufficient quantity of milk to last it any length of time, and therefore frequent

feeds will be necessary. Thus infants under 6 lb. require at least seven feeds in 24 hours, those from 6 to 10 lb. in weight six feeds in 24 hours, and those over 10 lb. five feeds in 24 hours. For the average infant of 6 lb. to 10 lb., therefore, it will be advisable to feed at 6, 9, 12, 3, 6, and 10 p.m., whereas it is usual to feed an infant over 10 lb. at 6, 10, 2, 6, and 10. Should the supply of breast-milk be too liberal, even in a small baby the intervals between the feeds must be lengthened. *The times of feeding should be rigidly adhered to and the infant wakened at these times conscientiously.* The mother should be careful to compress the nipples slightly shortly after the feed commences, to prevent the baby getting the milk too easily and gulping. The great bulk of the feed is obtained in the first five minutes, and 15 minutes ought to be sufficient time for the whole of the feed. At the end of the breast-feed the child should be wrapped tightly round in its shawl and placed in an upright position against the mother's shoulder, to allow him to break the wind. This should invariably be done after each breast-feed, as it is physiological for him to swallow wind, and an effort must be made to dislodge it, or discomfort and vomiting will result.

Diet of the nursing mother.—The mother's diet should be the one which agrees with her best in normal times, but in addition larger quantities of fluid, not necessarily milk, should be taken. If she can tolerate them, such sweet things as Horlick's Malted Milk or Ovaltine, with a high sugar-content, will be found to increase the flow of milk. It is quite wrong to supply the mother with gruel, stout and much milk, especially if these do not suit her normally. Fresh fruit and vegetables are essential. In treating constipation in the mother it is best to avoid salines and calomel, since they tend to lessen the quantity of milk. Senna, cascara or liquid paraffin will be found most efficient. It does not appear that foods which may be taken by the mother without ill-effect exert any ill effect upon the child. For instance, if the mother has eaten

tomatoes or oranges or grapes, and the following day a slight looseness appears in the infant's bowels, there would not appear to be any sound basis for attributing this to the mother's diet. Specifics disagreeing with the mother, however, are likely to cause an upset in the child.

Failure to breast-feed.—This may be due to a defect in the child or to a defect in the mother.

1. **Defect in the child.**—Anything which interferes with the strong suction of the infant at the breast will cause a failure in lactation. Nasal obstruction due to catarrh or adenoids, and hare-lip with cleft palate, cause great difficulty in sucking. Sometimes the infant is premature and under-weight, and not strong enough to suck properly. Or it may have congenital heart disease, or atelectasis of the lung. In these cases it may be necessary to pump off or express the milk and feed with a spoon or bottle. Some infants are poor suckers, the co-ordination between sucking, breathing and swallowing being badly developed. Success in such cases depends on the energy with which the situation is handled.

2. **Defect in the mother.**—This may be due to :

(i) Breast infections (abscess) ;
(ii) Cracked or sore nipples ;
(iii) Malformation of the nipples, that is, depressed nipples ;
(iv) Poorly developed breasts.

Where the breast has become infected so that there are tender areas in the breast substance or where there is actual abscess formation, breast-feeding may have to be discontinued entirely, since the general upset to the mother is considerable. In such circumstances the milk should be expressed or gently pumped off to relieve the mother, and the child fed on an artificial feed, or given milk from the uninfected breast with a complementary feed of cow's milk and water.

Methods of increasing the flow of breast-milk.—*Once lactation has been established, it is main-*

tained by the strong suction of the infant emptying the mother's breasts at regular intervals. This, then, is the most potent factor in the establishment and maintenance of lactation. The supply of breast-milk may be increased by :

1. Improving the eating and sleeping habits of the mother.
2. Seeing that she has a sufficient fluid intake and especially that she is having some of the malted foods. So-called galactagogues do not appear to increase the flow of breast-milk.
3. Making certain that the breast is completely emptied at each feed by pumping off or expressing the last drops of the breast contents.
4. Inducing in the mother a feeling of confidence in her ability to nurse her child successfully. This may demand an exaggerated air of optimism on the part of the doctor and nurse.
5. Breast massage and the application of hot and cold douches to the breast.

Underfeeding on the breast.—Authorities differ as to whether underfeeding or overfeeding is the more common in breast-fed babies. There can be very little doubt that of those breast-fed infants who are brought for medical advice, the vast majority are underfed.[1] The complaint is often merely that the child is failing to gain in weight, at other times that it is restless during the day and sleepless at night. The stools may be constipated when the supply is only very slightly deficient, but later they are small, slightly greenish and often frequent—so-called " hunger stools." Vomiting may also be a symptom. This is brought about by the child, having been put to the breast in a ravenous state, gulping what little milk there is, and with it much air. As the wind is returned some of the milk is returned with it, and this further reduces

[1] Donald Paterson and A. Marr-Geddes, " The Frequent Occurrence of Underfeeding in Early Infancy," *Arch. Dis. Child.*, Vol. 2, No. 11, Oct. 1927.

the already meagre supply of milk, so that at the next feed an even more ravenous infant gulps wind. It is this picture which has given rise to the expression "windy milk," though probably no such thing as "windy milk" exists—it is really a case of insufficient milk. An infant that is underfed cries a great deal, especially before its feeds are due. It also cries after being fed, due to colic from swallowed wind. This crying gives the parents the impression that the child has indigestion, and it is thought that the breast-milk is too rich or that the child is getting too much, and consequently water is often given before the feed to dilute the strength. If the infant is truly underfed, its condition will be aggravated by such treatment.

Diagnosis of underfeeding.—To ascertain whether an infant is underfed, a series of "test-feeds" should be given, that is, the infant should be weighed carefully, either naked or in its clothes or shawl, then put to the breast, then reweighed and the difference, which is the amount of milk obtained, carefully noted. A series of these tests taken over 24 hours is necessary in order to come to any proper conclusion, as it will be noted that the feeds vary in size. One is often told that plenty of breast-milk exists, and the mother insists that it flows away from her. The size of the breast, its tension, and the discomfort of the mother are taken as sure signs of the amount of milk available. None of these signs can be relied upon, the only certain criterion being a test-feed. Probably women doctors, mothers of difficult infants, are the hardest to convince of this.

Amount of breast-milk required in the day.—To be able to judge of the efficiency of breast-feeding it is necessary to know how much breast-milk a normal healthy infant requires to ensure that it will thrive. This will be found to be from $2\frac{1}{4}$ oz. to $2\frac{1}{2}$ oz. for each of the baby's pounds in the day, that is, a 6 lb. infant requires $6 \times 2\frac{1}{2} = 15$ oz. of breast-milk in the day. A 10 lb. infant requires $10 \times 2\frac{1}{2} = 25$ oz. of breast-milk in the day. It is easily seen, then, that one isolated

test-feed is of no value, but it must be the amount of breast-milk given during a particular day that is ascertained. Each infant is a law unto itself; some infants require slightly more food than others, and nervous highly-strung infants especially require more food than placid infants who sleep much. In cold weather more food is required than in hot, sultry weather, and therefore the figure given above is an average only.

Complementary feeds.—If it has been definitely ascertained by *test-feeds* that the supply of breast-milk is insufficient and at the same time that the infant is not thriving, then complementary feeds should be commenced. By "complementary feeds" is meant the giving of additional food at the end of each breast-feed, but not in place of a breast-feed. "Supplementary feeds" are bottle-feeds given in place of breast-feeds and are the first step towards weaning. In complementary feeding, therefore, both breasts should be given at each feed, so as to ensure that they are completely emptied. The breasts should be used in rotation, alternating the one to be used first at each feed. Immediately after the breast the bottle is given, with a sufficient quantity to make up the deficit. An example of this would be of an infant weighing 10 lb. and fed four-hourly—that is, getting five feeds in the day—who is obtaining from its mother 15 oz. of milk only. This child would be getting an average of about 3 oz. at each of the five feeds, but we know that an infant weighing 10 lb. ought to have $10 \times 2\frac{1}{2} = 25$ oz. in the day, which is an average of 5 oz. at each of the five feeds. Two ounces of a complementary feed should be offered to this child therefore at the end of each breast-feed.

Rules for complementary feeding.—1. The child should be left at the breast only long enough for the breast to be properly emptied, that is, if very little milk is present, for not more than three to six minutes. Sucking at the empty breast fills the infant with wind.

2. The complementary feed should be given after the breast-feed, and never before. This ensures that the breast is properly emptied.

3. Both breasts should be given at each feed, and they should be given in rotation.

4. The complementary feed should not be made very sweet, or the infant will refuse the breast and prefer the complementary feed.

5. Where possible, all the breast-feeds should be test-feeds and the deficiency made up to an exact quantity. It will probably be noted that the greatest deficiency is in the middle of the day and that therefore the largest complementary feeds will be required then.

6. Continuous efforts should be made to increase the supply of breast-milk (*see* p. 74).

Does the quality of breast-milk vary greatly?—It is difficult to get a good average specimen of breast-milk, as it necessitates the removal of a complete day's output. It is probable that the quality of breast-milk strikes a good average and that faults in it are rather of quantity than of quality. There can be no doubt that the vast majority of infants who fail to thrive on the breast do not owe their failure to a faulty quality of the breast-milk.

Overfeeding on the breast.—This is a fault not often seen by doctors, as Nature tends to correct the error herself.

Symptoms:

1. Frequent small vomits after feeds, not produced by eructations of wind.
2. Frequent loose motions. At first these are of a good colour, but later tend to contain curds and to burn the buttocks.
3. Colic, restlessness, disturbed sleep, failure to gain in weight. Very often the overfeeding is due to too-frequent feeding, or leaving the child at the breast for too long a period when there is an over-abundant supply of breast-milk.

Treatment:

1. Test-feeds should be done to ascertain the amount of breast-milk.
2. Only one breast should be given instead of both.
3. The periods between the feeds should be lengthened from three-hourly to four-hourly intervals and no night-feeds should be given.
4. The time at the breast should be shortened.
5. A little water may be given before the breast-feed.

Weaning and the commencement of mixed feeds.—Weaning should take place by degrees; thus the infant is not upset and the mother's milk is allowed to disappear gradually, preventing painful breasts. A period of cold rather than warm weather should be chosen. There is no set age at which weaning should take place. When an infant weighs 15 lb. it will be getting about 35 oz. of breast-milk from its mother, and it is seldom that the average woman can produce more than this without detriment to her own health. The solution of the difficulty is to give the child something besides breast-milk. *Mixed feeding* should commence at this point, usually about the sixth month. The first stage is the giving of one to two teaspoonfuls, increasing to two tablespoonfuls, of bone and vegetable broth (*see* p. 92) at the 2 p.m. feed. This may be liberally thickened with potato a little later. The second stage is the giving of a feed of groats, $\frac{1}{3}$ or $\frac{1}{2}$ teacupful, made with milk, at the 10 a.m. feed. This may be given from a bottle or, better, fed with a spoon. At 6 p.m. one-third or half a teacupful of some starchy preparation such as Groult's Cream of Rice, Robb's Biscuits, Neave's, Sister Laura's, or Ridge's Food, or Allenburys' No. 3 or Glaxo Malted Food may be given next, and forms the third stage. The fourth stage is the addition of one to two teaspoonfuls of the yolk of a soft-boiled egg to the groats at 10 a.m. Rusks should be given as soon as the lower incisors appear. Each of these stages should occupy about a week. At about nine months bottles may be substituted for

the breast, one feed at a time, and it will be found suitable to give a 7-oz. feed composed of 5 oz. cow's milk, 2 oz. of water and a heaped teaspoonful of sugar (*see* p. 90). Should the mother complain, the breasts may be relieved by supporting them with a bandage. Her bowels should be kept well open with aperients at this time, and the breasts should be emptied at bedtime by expressing.

Failure of the infant to thrive on normal breast-milk.—Normally the infant stool is acid, but some infants have an intestine which will not tolerate an acid stool. To make them thrive the stool must be alkaline. This may be brought about by the addition to the breast-milk of feeds high in protein. A practical means is to give one ounce of whole boiled cow's milk, with very little sugar added, in addition to the breast. Half may be given before the breast-feed and half at the end, from either a bottle or a spoon. Mead's " Casec " is also an excellent substitute for cow's milk ; or Albulactin, Skimmed Cow and Gate, Trufood or Glaxo may be used. In a certain very small number of cases the infant does not seem to tolerate even the smallest proportion of breast-milk. In these cases, which must be studied individually, weaning is to be advised.

ARTIFICIAL FEEDING

There are fundamental differences between cow's milk and breast-milk which can never be overcome. Breast-milk is received by the infant warm and sterile, while cow's milk reaches the infant after some hours and has always been contaminated. The protein and fat of cow's milk can never be made similar to that of breast-milk.

The main differences in composition between cow's milk and human milk have already been set out in Table XI, p. 70. The question arises whether it is necessary in artificial feeding that cow's milk should be modified to become as nearly like breast-milk as possible. The answer is that since we cannot produce a mixture like breast-milk we should concentrate on producing a

mixture on which the infant thrives as well as on breast-milk. This may necessitate departing widely from the breast-milk standard. Let us consider the nature of the components of cow's milk and their clinical importance. Cow's milk is composed of protein, (casein and lactalbumin), fat, carbohydrates and salts.

Protein.—When the protein of cow's milk reaches the stomach it is acted on by gastric secretions producing a clot. It is casein which clots becoming calcium caseinate. *These curds may be made smaller or modified by the following means:*

1. Boiling the milk precoagulates the casein, so that the curd is small when formed in the stomach. Pasteurization does not inhibit curd-formation.
2. Diluting the milk with water causes smaller curds which are more easily digested.
3. Dilution with cereal waters. On diluting the milk with a colloidal solution of barley- or oat-starch, the particles of casein seem to be held apart by this colloidal solution and the curds remain extremely small.
4. By peptonizing. The curd may be almost completely obliterated in this way, but the method is rather complicated and expensive for universal use.
5. By the use of dried and condensed milks. In the process of drying and condensing milks a change in the protein takes place, so that the curds are distinctly smaller than in fresh cow's milk.
6. By the use of alkalis. By alkalis are meant sodium bicarbonate, sodium citrate, calcium hydroxide (lime-water), magnesium hydroxide (milk of magnesia). Possibly these join up with the casein to form smaller molecules, but in any case the resultant curd formation is much smaller when such alkalis are added.

If there is an excess of protein the infant will fail to gain weight and tend to be constipated, since alkaline

constipated stools are produced by an excess of protein. Protein curds are seldom seen in the stools of infants, and when they are, they are large, bean-shaped, brown and semi-translucent. If underfeeding on protein occurs, the infant tends to be progressively anæmic and rachitic.

Fat.—This is probably the most difficult element to digest in cow's milk. The globules are large and split down into fatty acids which some infants are unable to assimilate. The stomach empties more slowly on high-fat foods, and in warm weather there is a tendency to diarrhœa. There is no method of modifying the fat of cow's milk.

Carbohydrate. Sugar.—This is present as lactose or milk sugar. Sugar is essential if a child is to gain weight, but very few infants can manage more than an ounce (8 teaspoonfuls) of sugar added to their day's feed. To metabolize the fat present in a feed it must be balanced by sugar to prevent ketosis (acidosis). The most easily assimilated sugar is dextrimaltose, not lactose, as is commonly supposed.

Excessive sugar-feeding causes:

1. A very rapid gain in weight, the infant retaining much fluid in its tissue and becoming extremely "watery."
2. A tendency to loose, frothy, acid stools, produced by the sugar breaking down into the lower fatty acids.

Insufficient sugar causes:

1. Failure to gain in weight; constipation.
2. Chronic biliousness and acidosis, with a dirty tongue.

The aim in infant feeding is to get a nice balance between the alkaline-producing protein and the acid-producing fat and sugar. The constipated child who fails to gain weight rapidly is an invitation to add more sugar to the diet. Some of the better-known forms of sugar available are Mead's Dextrimaltose, Wander's Nutromalt, and Mellin's Food. For practical purposes

the use of ordinary Demerara or cane sugar is quite sufficient in feeding the average healthy infant. Where there is a poor tolerance for sugar, however, dextri-maltose preparations will be found best.

Starch.—When an infant reaches the weight of about 15 lb. the carbohydrate must be increased along with the rest of its food, to keep the diet balanced. The average infant, however, is not able to manage large quantities of carbohydrate in the form of sugar, and it will be found beneficial to commence starches at this time. The starch, in process of digestion is split into sugar, but since this process is slow, fermentation is not so likely to occur. Many infants who fail to gain when sugar is added to their feed will gain rapidly when starch is substituted for a portion of the sugar. It is due to this fact that a number of the well-known starchy infant foods have gained their deserved reputations (*see* p. 67).

Water.—By diluting the feed it is made more digestible and supplies fluid which is utilized in the infants' metabolism. In summer more water is required. If water is withheld the infant becomes dehydrated, the urine is concentrated and scanty, and the temperature rises (*see* p. 127, Dehydration Fever). Water given in addition to breast-feeds helps to correct constipation.

Methods of Artificial Feeding

1. **Undiluted cow's milk.**—This will be found unsuitable in practice except in some few cases. The majority of infants will tolerate it, but it is not on the whole advisable.

2. **Humanized cow's milk mixtures.**—To make up a mixture roughly similar in composition to that of human milk sounds most attractive. It is well then, as the infant requires more and more food, to increase this mixture in amount only, but not in strength. Some normal healthy infants thrive well on this, but there are a large number who find the

quantity of sugar too great, and develop diarrhœa and flatulence. This is easily explained, as the total amount of sugar offered to the infant in the day when very young and small is but a small quantity. As the child increases in size and its demand for food increases, the total quantity of sugar offered in the day increases rapidly, and when the amount of added sugar in the day amounts to more than about an ounce, diarrhœa frequently results, especially in warm weather.

3. **Simple dilution of milk, with the addition of carbohydrate.**—The first step is to think in quantities necessary for 24 hours rather than in amounts necessary for individual feeds. The weight of the infant is much more help in calculating its food requirements than is its age, and while it may not lead to an absolutely accurate result it is the most useful basis we have at present. *The individual infant must be considered and is a law unto itself. No rule can be set out which covers every individual case.*

Calculation of the food requirements of infants.[1]—A breast-fed baby requires $2\frac{1}{2}$ oz. of milk per pound body-weight per day. It seems rational therefore that the artificial feed should be made up to the same quantity, that is a 10-lb. baby requires $10 \times 2\frac{1}{2} = 25$ oz. of fluid administered in the day.

Cow's milk requirements.—The milk should always be boiled before using, and it will be found that an infant requires $1\frac{1}{2}$ oz. of cow's milk for each of its pounds in the day. Thus an 8-lb. baby requires $8 \times 2\frac{1}{2} = 20$ oz. of fluid in the day; of this $8 \times 1\frac{1}{2} = 12$ oz. should be cow's milk and therefore 8 oz. of water will be added. Sugar of course must be added to this (*see* p. 85), and half a teaspoonful of cod-liver oil emulsion may be given three times daily. Put another way, the child requires a feed containing two parts of cow's milk to one of water. Give $2\frac{1}{2}$ oz. of this mixture with a level teaspoonful of sugar added for each pound of the body-weight in the day.

[1] Donald Paterson and Ruth Darby, "A Study in Infant Feeding—Food Requirements of Infants," *Lancet*, Jan. 31, 1925, p. 232.

Full-cream dried milk (Cow and Gate, Glaxo, Dorsella, Ambrosia, Lacta, Milkal, etc., *see* Table VIII).—One very heaped teaspoonful or measure of any of the above dissolved in one ounce of water reconstitutes one ounce of cow's milk. In calculating the food it will be found that the infant requires $1\frac{1}{2}$ measures or teaspoonfuls for each of its pounds in the day, that is a 7-lb. infant requires 11 or 12 heaped teaspoonfuls or measures of Glaxo spread over the day. The amount of water this would be dissolved in would of course be that quantity which it would get if it were being breast-fed, namely $2\frac{1}{2}$ oz. per pound body-weight per day. An example is an 8-lb. infant fed on Cow and Gate, who would get 12 heaped teaspoonfuls or measures of Cow and Gate dissolved in $8 \times 2\frac{1}{2} = 20$ oz. of water in the day. Sugar must of course be added to these feeds.

Sugar.—In giving both ordinary cow's-milk feeds and full-cream dried milk, sugar must be added. It will be found a very useful rule to add one level teaspoonful ($\frac{1}{2}$ dr.) for each of the infant's pounds in the day, that is, to the day's feed for a baby of 7 lb. 7 level teaspoonfuls should be added. To the 10-lb. infant's feed 10 level teaspoonfuls should be added. This infant, however, is fed five times in the day, so to each of his feeds two level teaspoonfuls or one heaped teaspoonful should be added.

Lactose is not the sugar of choice. The most easily absorbed sugar is dextrimaltose, a cheap form of which is Demerara sugar. The better forms are Mead's Dextrimaltose, Wander's Nutromalt, and Mellin's Food.

Humanized dried milks (New Glaxo, Allenburys' 1 and 2, Humanized Trufood, Recolac, etc., *see* Table X).

The essential thing about the humanized dried milks is that one very heaped teaspoonful or measure dissolved in 1 oz. of water reconstitutes as nearly as possible 1 oz. of breast-milk. In calculating the food requirements with these humanized dried milks one would give $2\frac{1}{2}$ heaped teaspoonfuls or measures dissolved in $2\frac{1}{2}$ oz. of water for each of the baby's pounds in the day. *No sugar need be added.*

The greatest use for humanized dried milks will be found in the first few weeks of life. Certainly after the third month it would appear desirable to increase the proportion of curd as the sugar is increased, and the change from humanized milks to full-cream mixtures at about this period is quite customary.

Lactic acid milk and its use.—This can be made in two ways. The first method, which as a rule is not a practicable one, is to add a culture of the lactic acid bacillus to cow's milk and incubate it for some hours at 55° F. At the end of 6 to 12 hours the desired degree of acidity is obtained. An easier and more practicable method is Marriott's method: To a pint of skimmed cow's milk, which has been boiled and allowed to cool, 45 drops of (B.P.) lactic acid are added drop by drop, stirring the while. Care must be taken not to warm it to any degree when giving it to the infant. Water and sugar must be added in suitable quantities.

The rationale of lactic acid milk feeding.—Milk has a buffer action, that is, it has an ability to absorb into itself either acids or alkalis without changing its reaction to litmus. Milk taken into the stomach therefore absorbs a large proportion of the available hydrochloric acid, leaving only a very small quantity free to raise the gastric contents to the optimum acidity for gastric digestion. In debilitated infants, especially those suffering from digestive disturbances, most of whom tend to have a hypochlorhydria, lactic acid milk feeding is of the utmost value. The milk is administered to the infant with a buffer already filled with lactic acid, therefore all the infant's hydrochloric acid is available to raise this feed to the optimum acidity for gastric digestion.[1] In various parts of the world, especially in warm climates, physicians are now making a routine of feeding babies, normal and otherwise, on this food and claim great success. It is undoubtedly of great use in feeding infants who are for any reason below normal.

[1] Lucy Wills and Donald Paterson, "A Study of Gastric Acidity," *Arch. Dis. Child.*, Vol. I, No. 4, p. 232.

Buttermilk.—Buttermilk is difficult to obtain fresh in this country, but an excellent dried buttermilk is now obtainable under the name of "Eledon," produced by the Nestlé and Anglo-Swiss Condensed Milk Company. This is in many respects superior to the fresh lactic acid milk, and is a highly successful food in cases of acute diarrhœa (*see* p. 123).

Fig. 8.—Soxhlet bottle and an ordinary boat-shaped bottle.

Choice of food.

1. **Unboiled cow's milk.**
   Advantages.—Unaltered taste, vitamins present, simple to prepare and cheap.
   Disadvantages.—Not sterile, not very digestible.
2. **Boiled cow's milk.**
   Advantages.—Cheap, digestible, sterile.
   Disadvantages.—Altered taste, partial destruction of vitamins.

3. **Dried Milks** (Full-cream Glaxo, Cow and Gate, etc.).
   Advantages.—Sterile, constant in quality, easily digested, easy to obtain, keep and make up.
   Disadvantages.—Expensive, slight deficiency of vitamins.
4. **Humanized dried milks** (New Glaxo, Recolac, Allenburys' 1 and 2, etc.).
   Advantages and disadvantages the same as those of full-cream dried milks.

Choice of bottle.—There are two types of bottle on the market—the boat-shaped bottle, such as the

Fig. 9.—Soxhlet apparatus. (*Messrs. John Bell and Croyden.*)

Allenburys' or Glaxo, and the upright or Soxhlet type (Fig. 8), with an opening at one end only. This latter has the advantage that it can be sterilized as it is stood on end, but it cannot be so readily kept clean as the boat-shaped bottle. The hole in the teat should be on the large rather than on the small side, as much more harm is done by the hole being too small, the infant becoming tired and tending to suck wind. The temperature of the feed should be about 100° F. Whichever type of bottle is used it will be found most convenient to make up the total feed for the day in the morning and to divide each feed out in separate bottles, which may then be brought to the boil by the use of a Soxhlet or some other convenient apparatus (Fig. 9).

Commencement of mixed feeding.—When a child weighs 15 lb., that is when it is aged about six months, it receives about 20-25 oz. of cow's milk in the day. This amount ought to be the maximum which any infant receives, and from this time on the amount should tend to decrease rather than increase. As the child requires more food, therefore, it should be added in the form of starches, since the full amount of carbohydrate as sugar, namely one ounce, has already been added. The feeds are four-hourly, and it will be found useful to feed as shown on p. 90.

This régime is arrived at by slow stages, each addition taking two to three days, and half amounts only being given until the child adjusts itself to the additions. For those children who find difficulty in managing the cereal a preliminary week on Benger's at 10 a.m. and 6 p.m. is a good intermediate stage. Some infants prefer the cereal as a semi-solid from a cup, well sweetened, with a little milk added. It is undoubtedly an advantage to push on with spoon-feeding at the earliest possible moment. When the mixed feeding is attempted as an addition to breast-milk, the administration of the food by spoon rather than by bottle is much more convenient. It is often an advantage to add some cow's milk to the soup.

Probably it is best to start by making the additions to the 10 a.m. feed, then to the 6 p.m. feed and finally to the 2 p.m. feed. The greater the variety of cereals fed, the more likely is success to be achieved. Wheat starch, such as in rusks and Robb's biscuits, is less rachitic than oat or barley starch, rice flour being intermediate. Oat flour should certainly not be given at more than one feed in the day.

MIXED FEEDING FOR BREAST-FED INFANTS FROM 6 TO 9 MONTHS OLD (WEIGHT 15 TO 18 LB.).

Feed baby at 6, 10, 2, 6, and 10 p.m.

6 a.m. Give both breasts, 7 minutes to each side.

10 a.m. Give one heaped teaspoonful of Robinson's Patent Groats or Patent Barley or Groult's Cream of Rice, with one

ounce of water and 2 oz. of boiled milk, sweetened with sugar. The cereal needs to be cooked for at least twenty minutes, or for an hour in a double saucepan.* A heaped teaspoonful of Allenburys' No. 3 or Glaxo Malted Food may be given instead of the other cereals. The feed may be given in a bottle or as a semi-solid from a cup.

After this feed give the breast.

2 p.m. Give two tablespoonfuls of bone and vegetable broth, with 1 oz. of water and 2 oz. of boiled milk, sweetened with sugar.

After this give the breast.

6 p.m. Give one heaped teaspoonful of one of the cereals mentioned in the directions for the 10 a.m. feed, with 1 oz. of water and 2 oz. of boiled milk, sweetened with sugar. *Do not give the same cereal as was given at* 10 *a.m.*

After this feed give the breast.

10 p.m. Give the breast only, as at 6 a.m.

N.B.—Orange or grape juice, two to three teaspoonfuls, diluted with water and sweetened with sugar, should be given daily. A convenient time for this is between 8 and 10 a.m., or at tea-time.

Give an eggspoonful of cod-liver oil emulsion immediately before three of the feeds.

Mixed Feeding for Infants from 6 to 9 Months old (Weight 15 to 18 lb.)

Feed baby at 6, 10, 2, 6 and 10 p.m. All milk should be boiled.

6 a.m. Give 5 oz. of milk, 2 oz. of water and one heaped teaspoonful of sugar.

10 a.m. Give 5 oz. of milk, 2 oz. of water and one heaped teaspoonful of sugar, to which had been added a heaped teaspoonful of either Robinson's Patent Groats or Patent Barley, or Groult's Cream of Rice. The cereal needs to be cooked for at least twenty minutes, or for an hour in a double saucepan.* A heaped teaspoonful of Allenburys' No. 3 or Glaxo Malted Food may be given instead of the other cereals. The feed may be given in a bottle or as a semi-solid from a cup.

2 p.m. Give 5 oz. of milk, one heaped teaspoonful of sugar and two tablespoonfuls of bone and vegetable broth.

* In making up the cereal for the 10 a.m. and the 6 p.m. feed, such as Groats or Cream of Rice, this may be added to the rest of the milk and water and the whole cooked for the specified time, or it may be cooked with water only and then stirred into the milk and water when it is thoroughly cooked.

6 p.m. Give 5 oz. of milk, 2 oz. of water and one heaped teaspoonful of sugar, to which has been added a heaped teaspoonful of one of the cereals mentioned in the directions for the 10 a.m. feed, but *not the same cereal* as was given at that feed.

10 p.m. Give the same as at 6 a.m.

N.B.—Orange or grape juice, two to three teaspoonfuls, diluted with water and sweetened with sugar, should be given daily. A convenient time for this is between 8 and 10 a.m., or at tea-time.

Give an eggspoonful of cod-liver oil emulsion immediately before three of the feeds.

Mixed Feeding for Children from 9 Months to 1 Year old (Weight 18 to 22 lb.)

Note.—The transition from the previous diet to this diet should be slow, taking, say, a fortnight.

On waking. Give one tablespoonful of orange juice, grape juice or grapefruit juice, diluted with water and sweetened with sugar. One rusk.

8 a.m. Give half a cup of cereal, such as Cream of Wheat, Wheatena, Groats, etc., with a spoon from a cup. A rusk and milk may be given instead of this, or half a Robb's Biscuit with milk.

Four mornings in the week, give half an egg, and two mornings toast crumbs or crisp toast fried in bacon fat.

The child should have 8 oz. of milk at this meal, to include that given with the cereal.

12.30 p.m. Give bone and vegetable broth, four tablespoonfuls with a little meat left on the bone and the soup thickened with potato and served with sieved green vegetables. As the child approaches its first birthday, the following may be added:—pounded chicken, sole, plaice, brains, or underdone scraped steak.

As a second course give Cream of Rice or tapioca or sago or semolina (Groult's), one tablespoonful with sugar, or jelly or apple sauce. The child should have 4 oz. of milk at this meal, used to make up the cereal or a junket.

4.30 to 5 p.m. Give Cream of Rice or tapioca or sago or semolina (Groult's). Instead of this a rusk and milk may be given or Robb's Biscuit and milk (half a cupful). The child should have 8 oz. of milk at this meal including that used with the cereal. Junket and stewed fruit or custard may take the place of the cereal food.

10 p.m. Give 4 oz. of milk if necessary, but the child should not be wakened for it. Normally the 10 p.m. bottle should cease between nine and ten months.

Directions for making bone and vegetable broth.

Take 1 lb. of veal bones or beef bones, well broken up.
Cover with water and add one teaspoonful of vinegar. Occasionally, say once a fortnight, add a piece of calves' or ox-liver (about 2 oz.).
Simmer for from four to seven hours.
Now add vegetables (one potato, carrots, cauliflower and green vegetables).
Simmer for one hour more, strain and allow to set.
Give one to two tablespoonfuls in the 2 p.m. feed.

The broth is best cooked in a double saucepan and should keep for three days if stored in a cool place.

Diet from 1 to 2 Years

On waking. The juice of half an orange, or a dessertspoonful of grape juice, diluted with water and sweetened with sugar. One small rusk.

Breakfast, 8 a.m. Give well-cooked Groats or Cream of Wheat or Wheatena (one tablespoonful) or rusks in milk or Robb's Biscuit in milk. In addition, toast crumbs fried in bacon fat, or a small rasher of crisp bacon, or half a soft-boiled egg with breadcrumbs (four days in the week) or pounded plaice or sole or fresh herring.
8 oz. of milk, to include that used with the cereal.

Dinner, 12.30 p.m. Fish, boiled or steamed, or pounded chicken or brains or sweetbreads or scraped raw or underdone steak or Irish stew. One heaped tablespoonful of boiled, baked or mashed potato. Sieved sprouts, cabbage, spinach or greens, cauliflower, carrot, parsnip or lettuce. These are all best steamed.
Milk pudding (Groult's Cream of Rice, tapioca, etc.), with stewed apples, prunes, junket or custard. Water to drink.

Tea-supper, 4.30 to 5 p.m. Rusks or Robb's Biscuit in milk, with a little honey, seedless jam, mashed over-ripe banana or custard or junket or stewed fruit, and a small piece of sponge cake. Pulled bread (zwieback).
8 oz. of milk, to include that used with the biscuit.

6.15 p.m. It ought not to be necessary to give anything after tea-supper. If the child can only eat a poor tea, however, some of the milk and rusks may be kept and offered before he is put to bed, but this should not be done as a routine.

NOTE.—Some of the solid constituents in this diet must be introduced slowly during the period from one to two years, the whole diet not being suitable to commence with. Red meat should be introduced slowly and in very small quantities at first.

DIET FROM 2 TO 5 YEARS

On waking. Orange, grape or grapefruit juice, diluted with water and sweetened with sugar.

Breakfast, 8 a.m. Cereal, such as porridge, Cornflakes, Grapenuts, Shredded Wheat, Cream of Wheat or Wheatena, or in place of this stewed apples, prunes or figs.

In addition three mornings in the week an egg, three mornings rashers of crisply-fried bacon, one morning fish. Crisp toast or rusk spread with butter. 8 oz. of milk, to include that given with the cereal.

Dinner, 12.30 p.m. Cutlet, mince or stew, liver, underdone beef or steak, finely cut up, or brains, fish, pounded chicken or sweetbreads. Red meat four days in the week, white meat three days. One level tablespoonful of any of these.

One tablespoonful of boiled, baked or mashed potato, and one of finely-mashed carrots, cauliflower, sprouts, spinach or greens.

Milk pudding or stewed fruit, one heaped tablespoonful.

Water to drink.

Tea-supper, 4.30 to 5 p.m. Crisply-toasted wholemeal or brown bread, pulled bread (zwieback) or thin bread and butter or rusks and butter, or cream cheese sandwiches, mashed over-ripe banana or stewed fruit, seedless jam or jelly or honey. A small piece of sponge-cake may be added. 8 oz. of milk.

6.30 p.m. If the child has left some of his tea, the remnants may be offered at 6.30, but it will be found best to give nothing before going to bed.

NOTE.—As the child approaches the age of 5, larger helpings of food (except milk and eggs) may be given. Sweets such as barley-sugar and plain boiled sweets may be given immediately after dinner and tea.

Groult's Cream of Rice, tapioca, sago and semolina will be found most suitable for children.

Diet for Children of School Age

The growing child requires a substantial and mixed diet, It should contain fresh meat, an abundance of fresh vegetables and fruit, butter, milk and occasionally eggs, and should be properly balanced with sugar and starch. It should be remembered that the child requires much more food relative to its size than does the adult.

Breakfast. Either on waking or to commence breakfast, fresh fruit is most advisable. A ripe apple, orange juice, a few grapes or half a grapefruit should be given.

Cereals should be given after this, especially during the winter months. Porridge, Grapenuts, Shredded Wheat, Force, Puffed Rice, Wheatena, Cream of Wheat, Groats, Creamed Barley are all suitable. A small helping should be given, so that the child's appetite is not satisfied.

Three mornings in the week give an egg, soft-boiled, scrambled or fried, with or without bacon. On the other mornings give crisply-done, thin rashers of bacon with tomatoes, or fish (sole, plaice, fresh herring or kipper). Liver may be given occasionally. Crisp toast, butter and marmalade should finish the meal.

After the age of nine or ten years sausages may be introduced into the diet.

Drinks. A glass of milk or weak tea and milk.

Dinner. Cutlets, fresh mince, a cut of roast of beef, steak, mutton, on four days in the week, and chicken, fish, sweetbreads, liver or kidney on three days. Boiled, baked or mashed potato, with carrots, cauliflower, parsnips, or green vegetables, such as spinach, sprouts or cabbage, or peas or beans. Green salads.

For older children, rabbit may be given occasionally.

As a second course give either stewed fruit or milk puddings, or custard or sponge puddings. Suet pudding should not be given before the age of ten years. Water to drink.

Tea-supper. (*See* note below for directions when tea and supper are given separately.)

Thin bread and butter, with fruit, such as baked apple, or stewed prunes or mashed over-ripe bananas, or milk pudding or honey, jam or jelly, or cream cheese sandwiches. Sponge cakes. For drinks milk or weak tea with milk.

Note.—As the child approaches the age of seven or eight, a more and more substantial tea-supper is required. Occasionally

an egg may be introduced, or vegetable broth, or fish or macaroni or spaghetti, or tomatoes fried with breadcrumbs or cream cheese, or a milk pudding may be added to the meal. At the age of about nine to ten, however, it is best to give a very light tea at 4.30, and supper should be instituted at 6 to 6.30, the child going to bed at 8.

Milk.—Owing to the large number of children who cannot tolerate much milk, a good average per child is ¾ pint per day. In exceptional cases where it is well tolerated, more may be given with advantage, but it should not in any circumstances be pressed.

CHAPTER IV

DISEASES OF NUTRITION

Composition of normal stools.[1]—Normal breast fed infants at first pass stools consisting of meconium and debris from the intestinal tract. These stools are greenish-brown in colour and loose. After the fourth or fifth day the stool commences to have a more yellow colour as the breast-milk appears. Such stools are often described as having a mustard colour and their reaction is acid. In artificially-fed infants, the colour of the stool largely depends on the type of food given. They are loose, frequent in number and may be formed or almost formed. If the proportion of protein in the feed is high they are more formed and tend to have a cheesy or offensive odour; they are alkaline to litmus. Malted foods tend to produce a brown stool, and buttermilk mixtures produce an olive-green stool with a characteristic odour.

Appearance of abnormal stools.—For the purposes of examination the stools should first be broken up and pressed out so that the interior may be seen, as stools which have stood for some time occasionally exhibit a bright green exterior, with a normal yellow interior.

Grey stools are due to an excess of fat and are characteristic of the older child who is liverish or bilious, the fat covering the normal yellowish-green bile.

White stools may be seen when either a very large quantity of fat is present or the bile is deficient. In congenital obliteration of the bile-ducts the stools remain white. A good example of excess of fat in the stool is

[1] Donald Paterson, "Normal and Abnormal Stools in Infants," *Maternity and Child Welfare*, July 1923.

seen in cœliac disease, where the motions are large, porridge-like and putty-coloured.

Black stools are the result of the presence of either bismuth, iron or charcoal, or they may denote the presence of blood, when the stools may be termed tarry. In melæna neonatorum such stools are characteristic.

Green stools suggest that there is an increase of peristalsis, hurrying the normal green bile through the bowel before it can be oxidized into a yellow-coloured bile-pigment. In acute infections of the bowel with diarrhœa such stools are constantly found. Accompanying the partially digested food there is often mucus present and occasionally there may be blood.

Green stools are also seen where the damage to the bowel is due to fatty acids from overfeeding on fat or carbohydrate. In such cases the buttocks are also burnt. The starvation stool is green merely because it consists almost entirely of bile. These are actually seen in congenital pyloric stenosis.

Unusual ingredients and their meaning.—Stools often contain some of the following:

(1) *Blood*, which often accompanies a formed motion in a fat child, and is due to an anal fissure. It may also be present in melæna neonatorum. Hæmorrhoids are not uncommon in children and may give rise to bleeding. Accompanying intussusception, the "red-currant jelly" motion is characteristic.

(2) *Mucus* suggests that there is catarrh of the bowel, and when it appears on the outer surface of the stool only it denotes colitis rather than enteritis, as in the latter the mucus is intimately mixed with the stool. Much mucus is passed after a dose of castor oil, which causes acute irritation of the bowel wall.

(3) *Pus* is present when there has been inflammation of the bowel-wall, such as is found in true typhoid-dysentery infections.

(4) *Curds* are the most common abnormal constituents of the stool and are almost always composed of fat. They should dissolve in ether and will float on water. It is very seldom that curds are due to

Fig. 10.—Severe rickets, showing enlargement of the wrists and ankles and curving of the tibiæ. (*By courtesy of Dr. Hector Cameron.*)

protein, especially if the milk has been boiled. When this is the case, however, the curds have a characteristic appearance, being large, square and Lima-bean-like.

For a description of worms which may appear in the stool *see* p. 472.

Reaction of stools.—The stools of breast-fed infants are acid normally, those of bottle-fed infants are alkaline. When, however, in the artificially-fed infant an excess of fat or sugar is present the stools may become markedly acid, due to the presence of fatty acids.

DISORDERS DUE TO WRONG FEEDING

Rickets

Probably no disease in infancy is so widespread as rickets, and especially may this be said of countries as far north as the British Isles.

Age-incidence.—The most active signs of rickets appear between the ages of six months and two years, but there seems no reasonable doubt, however, that rickets is in process of development almost from birth onward. The florid manifestations would appear to be held off for at least three months and to be uncommon before six months.

Clinical picture. Bony manifestations of rickets.—The head is massive and large. The teeth are late, often none appearing until after one year of age, and when they do come the enamel is poor and caries takes place early. The bones of the legs and arms bend with pressure, so that the tibiæ and femora are bent either in or out, giving rise to knock-knee or bow-legs. The forearms are curved, due to the child supporting its weight when in a sitting posture (Fig. 10). The anterior fontanelle is normally closed at about one year to 15 months. In rickets it remains widely open even up to the second year.

Deformities of the chest.—Harrison's sulcus is a groove running round the chest at the attachment of the diaphragm, the pull of the diaphragm on the soft ribs having drawn them in at this point, causing the

splaying out or eversion of the lower edges of the ribs. At the costo-chondral junction beading is found, and these enlargements are as prominent on the inside of the rib as on the exterior. The back is bent, either a kyphosis or

Fig. 11.—Severe rickets, showing the typical chest of the greyhound, large abdomen and square philosopher's head. (*By courtesy of Dr. Hector Cameron.*)

scoliosis being present. This is due to muscular weakness, however, as the curve is easily undone if the child be picked up beneath the arms or placed on its face.

As a rule, during the active stage of rickets pallor and anæmia are common, but there are exceptions to this. Sweating of the head is a constant accompaniment and may be most striking. The muscles generally are

soft, flabby and diminished. There is a tendency to indigestion and catarrh of the stomach and bowels. *The old saying that the rickety child has the head of a philosopher, the chest of a greyhound, the legs of a grand piano and the "tummy" of a poisoned pup, is extremely apt* (Fig. 11).

Craniotabes.—In the young infant, accompanying other manifestations of rickets there is sometimes gross thinning of the skull-bones, so that on slight pressure they give way with a feeling like pressing on parchment. This is known as craniotabes, and premature infants and the poor one of twins often suffer in this way.

Fig. 12.—Carpopedal spasm in tetany.

Nervous manifestations of rickets. — Hyperexcitability of the nervous system, giving rise to certain symptoms, may appear in a case of acute rickets.

Tetany (spasmophilia).—Tetany manifests itself by—

1. *Facial irritability* or Chvostek's sign.—By tapping over the cheek, so that the facial nerve is irritated the muscles innervated by this nerve constantly pass into spasm. The whole face gives a well-marked twitch on that side.

2. *Carpopedal spasm* or Trousseau's sign.—The hands are held in what may be termed the accoucheur's position, with the fingers fully extended, but slightly flexed on the palm, and the thumb adducted (Fig. 12).

The toes assume much the same position as the hands. There seems no doubt that the child is in pain and suffers from " cramps " of the hands and feet. Where the hands are not in the typical position, this may be brought about in mild cases of tetany by gentle pressure on the forearm of the child, so that the hand becomes congested.

3. *Laryngismus stridulus* (crowing). A spasm of the larynx occurs whenever the child is suddenly roused, frightened, crossed or annoyed. The breath is held, the face becoming blue, much as during a whoop in whooping-cough, then it relaxes and a long-drawn inspiratory cry is heard. This is a dangerous condition and sometimes proves fatal.

4. *Convulsions* (*see* p. 347).—Children with acute rickets and tetany are very prone to convulsions, which often occur so frequently that the child passes from one convulsion to another. There is no way by which such convulsions can be distinguished from early epileptic fits except by noting the presence of other manifestations of active rickets.

Etiology.—Nervous rickets or *spasmophilia* is characterized by a low blood-calcium. Normally this should be about 10 mgm. per cent. In *bony rickets* the blood-phosphorus is deficient, being less than the normal, which is 5 mgm. per cent. In turn the calcification of the bones is deficient, so that there is actually less calcium in the bones than is the case in the normal child. Some of the factors bringing this about are :

1. *Lack of the fat-soluble vitamin* (*vitamin D*) which promotes growth and deposition of calcium.—This vitamin is commonly found in animal oils, such as cod-liver oil, cream, butter and egg-yolk, and to a less extent in beef and other animal fats. A diet low in animal protein and fat and high in farinaceous foods, therefore, will tend to produce rickets. Since this is characteristic of the diet of the poor, rickets is most commonly found among poor people. Milk produced by stall-fed cattle in winter-time is often grossly deficient in the vitamin.

2. *Lack of sunshine* (confinement, with lack of fresh air and exercise).—The rays of the sun, by acting on the skin and the bloodstream, have the power to produce the fat-soluble vitamin in the patient and to promote calcium deposition just as efficiently as if vitamins are given in the food. A vitamin-deficient diet in the presence of sunshine might be tolerated, but the same diet in the absence of sunshine would produce gross rickets.

3. *Calcium deprivation.*—Even in the presence of sufficient vitamin and sunshine, if the diet is so poor in calcium that too little of this mineral is available for the needs of the body, rickets will result. Thus foods such as some of the sweetened condensed milks, where the protein and fat are extremely low and the carbohydrates unduly high, may be actually deficient in calcium. Some of the farinaceous proprietary foods administered with too little milk will also tend to produce rickets in this way.

Pathology.—Normally at the epiphysial line, each cell gives rise directly to another, and that in turn gives rise to another, so that the line remains a straight one. In rickets the epiphysial line is wavy and irregular. One cell may give rise to two or more, so that there is a tendency for a widening of the cartilaginous area at the epiphysis. This is seen clinically by the gradual appearance of cartilaginous beads on the ribs and cartilaginous enlargements at the wrist and ankle. Being soft and cartilaginous the bones are easily bent or deformed. It is difficult to say why the calcium is not laid down in the bones. Often the diet contains sufficient calcium, but the element remains unabsorbed, and thus fails to reach the bloodstream and take its place in the formation of bone. Vitamins increase the flow of calcium from the bowel to the bloodstream and cause the deposition of the calcium present in the blood in the growing bone.

Diagnosis.—The clinical diagnosis of rickets is extremely difficult in slight cases. Since the bony malformations remain long after the condition has

healed, the clinician is apt to diagnose rickets in those cases where it is already cured. If nervous manifestations are present in addition, however, we may be reasonably sure that the condition is active. An examination of the blood-calcium or -phosphorus is difficult and expensive. There is no way so certain as by examining the epiphyses with the X-rays, and a radiogram of the wrist shows clearly whether the child has rickets (Plate 1).

Prognosis.—Once the bony deformities are present in marked degree, they remain to a greater or less extent throughout life. If the rickets has been allowed to continue, stunting and dwarfing tends to occur. The nervous manifestations, however, leave no residuum, so far as is known. The prognosis as to life is excellent, though death occasionally results from laryngismus stridulus or from some acute infection engrafted on florid rickets. Rachitic infants withstand infection extremely badly.

Treatment. 1. *Dietetic.*—The diet should contain a sufficient quantity of animal protein and fat, and any excess of farinaceous food should be done away with; in other words, the diet should be well-balanced. Up to nine months the infant should have from half to one pint of milk as a minimum. After this age eggs and bacon fat, and later fresh meat, fish and chicken should be introduced. (*See* pp. 90-95).

2. *Medicinal.*—Nothing is so potent in the prevention or cure of rickets as cod-liver oil. Every infant, breast-fed or artificially-fed, should be given cod-liver oil as a prophylactic, but in rickets once developed, this is a necessity in every case. From half to one teaspoonful of an emulsion given three times daily should suffice—that is, from 15 to 30 drops of the crude cod-liver oil three times daily. One of the concentrated irradiated ergosterol preparations, such as Ostelin, Vigantol, Viosterol, or Radiostol, may be used with advantage. Usually one or two drops three times daily are necessary. Where *tetany* is present it is well to give from 3 to 5 grains of calcium chloride three times

Plate 1.—Radiogram of wrist in a case of severe rickets.

daily for some weeks, a larger dose being necessary for the first few days.

3. *Hygienic.*—Sunshine (artificial or real), fresh air, exercise, all tend to take the place of cod-liver oil. Massage and artificial sunshine, with exposure of the limbs to fresh moving air as far as possible, should be given in the winter months in the British Isles.

Vitamins.—Vitamins are those substances the absence of which gives rise to certain diseases. Such conditions have on this account been named "deficiency diseases."

Vitamin A, or fat-soluble A, would appear to be necessary for normal growth. Its absence leads to the disease keratomalacia and to an increased susceptibility to bacterial infections, more especially of the bronchi and intestines.[1, 2] This vitamin is found dissolved in animal fats, but is absent from vegetable oils. It is present in large amounts in cod-liver oil, butter, milk and yolk of egg. Milk which is just brought to the boil or scalded still contains the vitamin, but in pasteurized milk, or that which has been heated to a lower temperature for a long time, this vitamin tends to be destroyed. In both condensed and dried milks vitamin A is present to a greater or less degree.

Vitamin B, or water-soluble B, is necessary for normal growth. This vitamin now appears to be divisible into two groups,—an anti-neuritic factor, the absence of which leads to beriberi or polyneuritis, and an anti-pellagra factor, the absence of which leads, certainly in rats, to a syndrome resembling pellagra. Vitamin B is found in meat, vegetables and milk, but also abundantly in yeast preparations, such as Marmite and Fleischmann's Yeast. It is also present in Bemax and Vitmar.

Vitamin C, or water-soluble C, is the antiscorbutic vitamin, the absence of which from the diet results

[1] H. N. Green and E. Mellanby, "Vitamin A as an Anti-infective Agent," *Brit. Med. Journ.*, No. 3,537, Oct. 20, 1928, p. 691.

[2] C. E. Bloch, "Diseases Arising in Consequence of a Deficiency in the Fat-soluble A Factor," *Am. Journ. Dis. Child.*, Dec. 1924, Vol. 28, pp. 659-667.

in scurvy. This vitamin is found most abundantly in fresh vegetables and especially fresh fruit, such as orange, grapefruit, grape juice and tomato juice. It is destroyed by heating, but in the ordinary preparation of food, and also of dried milks, it is only partially destroyed, quite sufficient being left to protect against scurvy, provided fresh fruit juice is added to the daily diet. There are certain infant foods, however, prepared in such a way that this vitamin is entirely destroyed, and unless, as directed on the tins, extra fruit juice is given, scurvy rapidly develops. This is of great practical importance. *Wherever an infant is fed artificially it should be given fresh fruit juice, two to three teaspoonfuls daily.*

Vitamin D, or fat-soluble D, is the antirachitic vitamin. It is the only vitamin of whose chemical composition something is known. The substance ergosterol, when exposed to ultra-violet rays, becomes endowed with antirachitic properties. Vitamin D is usually found in association with fat-soluble A. If vitamin D is withheld, growing animals develop rickets, and there is a deficient calcification of both the bones and teeth. Some of the best-known sources of this vitamin are milk, cream and butter, especially from cows that have fed on green pasturage and in sunny climates. The yolk of eggs is rich in this vitamin also, but the most abundant source is cod-liver oil. There are concentrated forms of vitamins A and D on the market sold under such names as Radiostoleum, Radiomalt, Vitamalt, Ostelin, Ostomalt, Vigantol, Viosterol, etc.

Infantile Scurvy (Barlow's Disease)

This is a deficiency disease, due to an absence of the water-soluble C vitamin—that is, the vitamin which is most commonly found in fresh vegetables, fruit and meat.

Age-incidence.—Scurvy is commonly found between the ages of six months and one year, in artificially-fed babies, especially those fed on proprietary foods.

Etiology.—The process of manufacture of some of the proprietary foods apparently destroys the water-soluble C vitamin, with the result that at the age of about six or seven months scurvy develops. Cases of scurvy are also occasionally seen in infants fed on milk which has been pasteurized or boiled. Undoubtedly coincident infections tend to precipitate and augment an attack of scurvy.

Pathology.—Due to the deficiency of this vitamin C

Fig. 13.—Case of scurvy, showing orbital hæmorrhages.

there are universal hæmorrhages into the growing ends of the bone, into the gums, kidney, costo-chondral junction, beneath the periosteum, into the bone-marrow itself and subcutaneously, especially about the eyes (Fig. 13) ; also into the joints.

Symptoms.—The mother notices that the infant is unhappy, especially when anyone approaches it and attempts to pick it up. When left perfectly still the child seems less anxious. Moving it, therefore, is painful. The limbs are kept still and the child appears pale and fails to thrive. Should treatment be with-

held, purpura next appears on the skin, especially about the eyelids. An examination of the blood shows the platelets to be quite normal. Swellings appear over the shins, knees or thighs, and X-rays show these to be large subperiosteal hæmorrhages (Plate 2). The urine is red or smoky, and a microscopic examination shows this to be due to the presence of red blood-corpuscles. Blood is always present in the urine of scorbutic patients, sometimes in microscopic amounts only. At this age there are usually two lower incisors, and about the base

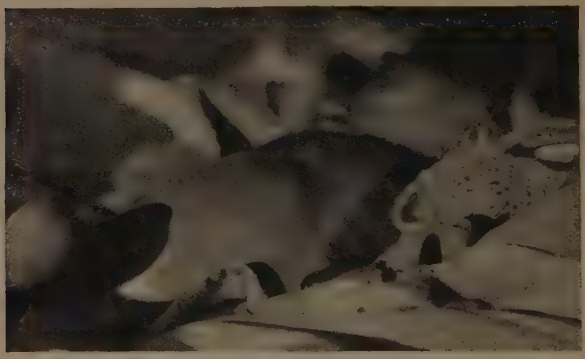

Fig. 14.—Case of scurvy, showing beading of the ribs.

of these will be seen purple hæmorrhagic areas, and the gums are swollen. An examination of the ribs shows that the sternum has become depressed and dislocated at the costo-chondral junction—that is, passing the hand out from the sternum towards the ribs the depression is reached, then the sharp tip of the rib is felt. This is quite unlike the beading in rickets, which is a smooth oval beading, with slight depression at either side, and not only at the sternal side, as in scurvy (Fig. 14).

Diagnosis.—Children who appear moderately well nourished and cry incessantly may be thought to be suffering from acute osteomyelitis, especially if the swellings are large and accompanied by some infection

Plate 2.—Radiogram in a case of infantile scurvy showing a subperiosteal hæmorrhage at the lower end of the femur.

(By courtesy of Dr. Hector Cameron.)

producing a fever. If the purpura is extensive the case may be thought to be one simply of purpura hæmorrhagica, or if one limb is specially involved, scurvy may be mistaken for traumatic dislocation of the epiphysis or a fracture or sarcoma. Where the hæmorrhages are not very apparent, such diagnoses as teething or earache are commonly made. The clinical picture, however, is a very definite one: hæmorrhages about the gums, tenderness of the limbs with or without swellings, blood in the urine, backward dislocation of the sternum at the costo-chondral junction, and hæmorrhage beneath the periosteum, the hæmorrhages being confirmed by X-rays.

Prognosis.—Properly treated, all cases of scurvy should recover completely. When the case is complicated by some infection, scorbutic infants resist the infection badly and the prognosis becomes less favourable.

Treatment.—The first step is to take the child off its scorbutic diet. Where it has been fed on a proprietary food which has been added to cow's milk, the proprietary food should be removed and cow's milk, water and sugar alone given for a time. Vitamins in the form of orange juice, grape juice, grapefruit juice, or tomato juice, should be given. Quantities of the latter equivalent to from one to two tablespoonfuls ought to be given per diem, diluted with water and well sweetened with sugar. If the bowels tend to be a little loose, this is no contra-indication. Potato-cream, suggested by Dr. Still, made by adding the floury portion of the potato immediately beneath the skin to cow's milk, to the consistency of cream, is most useful. Six to eight teaspoonfuls of this should be given during the day. Tinned tomato juice may be used if none of the above-mentioned fruits is available.

Prophylaxis.—All artificially-fed infants from a fortnight old should be given fruit juice daily, two to four teaspoonfuls of orange, grape or grapefruit juice being given in a bottle, diluted with water and well sweetened with sugar. With such an antiscorbutic added it is immaterial what food is then used, as scurvy cannot develop.

Cœliac Disease (Herter's Infantilism, or Intestinal Infantilism)

This may be defined as an inability on the part of a child to absorb and utilize fat, although digesting it apparently normally.

Sex-incidence.—The disease is found three times more commonly in girls than in boys.

Age-incidence.—Cœliac disease is rarely seen before the first year, and usually develops between the ages of one and two years.

Symptoms and diagnosis. — The complaint is usually that the child is failing to thrive and is passing large, pale-coloured motions. They are often referred to as "putty-like." They are very offensive in odour, and at times constipated; at other times they are loose, frothy or bubbly. During the bouts of diarrhœa weight is lost rapidly. Marked infantilism is a feature, a child of 2 or even 3 years weighing only 15 to 20 lb. and being proportionately small in size. The abdomen is large and the rest of the body wasted. On palpation the liver is not felt, and on percussion is found to be extremely small. The buttocks are wasted. A diagnosis of tuberculous peritonitis has often been made, but careful palpation of the abdomen reveals the fact that no large masses can be made out. Accompanying the bouts of diarrhœa, attacks of acute tetany occur at times. The bones are thin and show osteoporosis when examined by X-rays.[1] Active rickets occurs in some cases when recovery is taking place (Figs. 15 and 16).

Etiology and pathology.—It is considered by some that there is an initial infection by an organism such as the dysentery bacillus. Others contend that no such affection as cœliac disease exists, but that this clinical picture is merely the end-result of a chronic overfeeding on fats from birth, so that a gross intolerance

[1] L. G. Parsons, "The Bone Changes Occurring in Renal and Cœliac Infantilism and their Relation to Rickets," *Arch. Dis. Child.*, 1927 Vol. 2, pp. 1–25.

Fig. 15.—Case of cœliac disease.

Fig. 16.—Three cases of coeliac disease of the same age, with one normally grown child (second from right) of the same age.

of fat has been brought about. A post-mortem examination reveals the fact that the bowel, although thin, shows no other lesion. The liver is small (Fig. 17), but microscopically normal, and no other organs show abnormality. Biochemically the stools contain an excess of fat, but this has been well split. Of a normal dried stool, 25 per cent. ought to be fat; in cœliac disease 75 to 90 per cent. is common. About 75 per cent. of the fat present

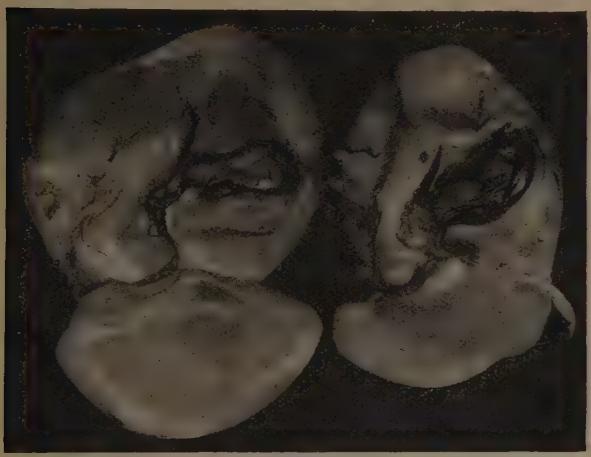

Fig. 17.—Liver in cœliac disease. The larger of the two is that of a normal child of 18 months, the weight being 18 oz. The smaller is that of a case of cœliac disease of the same age, the weight being only 9 oz.

ought to be split; this is the case in cœliac disease. Clinically this disease in many features is extraordinarily similar to adult sprue.

Prognosis.—Cœliac disease is much commoner among children of the well-to-do than among the poor, so that facilities for treating such cases are as a rule available. The mortality, which was formerly comparatively high, has fallen greatly, and with care and understanding the disease is now most amenable to

treatment. There is a tendency for the infantilism to persist, the children being undersized and rather stocky in form, even in adult life ; but as a rule the condition passes off more or less completely at the age of six or seven.

Treatment.—Since fats are not absorbed and tend to produce fatty acids and looseness in the child, the diet should be as nearly fat-free as possible. Protein does not disagree and a large amount should be given. Articles well tolerated are scraped raw or underdone steak, pounded chicken, fish, and broths from which the fat has been skimmed. Milk should be given as skimmed dried milk, and only in small amounts. Buttermilk or skimmed lactic acid milk may be substituted for this if the child does not like the dried milk. Vegetables are best given after being put through a fine sieve, and green vegetables, which are bulky and tend to form gas, should be avoided. Potato, cauliflower, parsnip, turnip may all be given after careful preparation. Rice pudding, of which the rice has been boiled and the water removed three times, is found most suitable. Probably the best preparation is Groult's Cream of Rice, sold in England by Lazenby and Co. Cereals such as groats or porridge, or some of the patent cereals, should be given in small or moderate amounts only.

Eggs, especially the yolk, should be avoided, the white only being used at first, gradually adding one teaspoonful of the yolk. Bread should take the form of rusks or crisp, dry toast. Cream should not be given and butter only very sparingly. It is well to give a few drops of cod-liver oil emulsion three times daily, since all the active fat has been removed from the diet, and in this way the growth factor is supplied. Artificial sunshine is useful. In some cases over-ripe bananas have proved most beneficial, but they should be cautiously admitted in each case. When this food is well managed the progress made is dramatic. Castor oil is useful as a purge in these cases, since it tends to leave the child constipated afterwards. Purges which tend to cause

continuous looseness of the bowels should be strictly avoided.

The following table shows a week's suitable diet for a case of this kind :

TABLE XII

Diet in Cœliac Disease (Age 1 to 2 Years)

On waking.	*Breakfast.*	*Dinner.*	*Tea.*
Monday.			
Orange juice, 1 rusk.	1 tablesp'ful Force, 1½ rashers bacon, 1 round dried bread with honey. Skimmed milk, 4 oz.	1½ tablesp'fuls chicken, 1 potato, 1 tablesp'ful sieved cauliflower. Milk jelly, 4 oz.	2 rusks with ½ banana (sieved) and honey, 1 sponge - cake. Skimmed milk, 4 oz.
Tuesday.			
Grape juice, 2 Mellin's biscuits.	Groats 4 oz. (milk 2 oz., water 2 oz.), 1 round dried bread with butter and honey, ½ rusk. Skimmed milk, 4 oz.	1½ tablesp'fuls raw beef, 1 potato, 1 tablesp'ful sieved cabbage. Cream of rice, 4 oz.	2 rusks, ½ banana and honey, 1 sponge biscuit, 1 Mellin's biscuit. Skimmed milk, 4 oz.
Wednesday.			
Orange juice, 2 Mellin's biscuits.	1 tablesp'ful Force, 1 tablesp'ful pounded mutton, 1 round dried bread with butter and honey. Skimmed milk, 4 oz.	1½ tablesp'fuls chicken, 1 potato, 1 tablesp'ful sieved cauliflower. Baked apple with 1 tablesp'ful custard.	2 rusks, ½ banana and honey, 1 sponge - cake, 1 biscuit. Skimmed milk, 4 oz.
Thursday.			
Prune juice, 2 Mellin's biscuits.	1 tablesp'ful Force, 1 egg (1 teasp'ful yolk), 1 round dried bread with butter and honey. Skimmed milk, 4 oz.	1½ tablesp'fuls raw beef, 1 potato, 1 tablesp'ful sieved sprouts. Sago, 4 oz.	2 rusks, butter and honey, 1 biscuit, 1 sponge-cake. Skimmed milk, 3 oz.
Friday.			
Orange juice, 2 Mellin's biscuits.	1 tablesp'ful Force, 1½ rashers bacon, 1 round dried bread with honey, 1 Mellin's biscuit. Skimmed milk, 4 oz.	1½ tablesp'ful sweetbread, 1 potato, 1 tablesp'ful sieved cauliflower. 3-times-boiled rice, 3 oz.	2 rusks, ½ banana and honey, 1 sponge-cake, 1 biscuit. Skimmed milk, 4 oz.
Saturday.			
Grape juice, 2 Mellin's biscuits.	1 tablesp'ful Force, 1 tablesp'ful sweetbread, 1 round dried bread with butter and honey, 1 Mellin's biscuit. Skimmed milk, 4 oz.	1½ tablesp'ful raw beef, 1 potato, 1 sp'ful sieved cabbage. Junket, 4 oz.	2 rusks, ¾ banana, honey, 1 sponge-cake, 2 biscuits. Skimmed milk, 4 oz.

DISTURBANCES OF DIGESTION

Summer Diarrhœa (Epidemic Diarrhœa)

Summer diarrhœa, *i.e.*, diarrhœa during the summer months, is a term which may embrace several different conditions. Various groups of cases are covered, the etiology of which differs, so that an infant with an acute fat dyspepsia or some parenteral infection may come under this heading quite as appropriately as a case of Flexner, Shiga or Y-dysentery. The author prefers a more explicit classification, and the following is both easy and generally accepted:

1. *Infective diarrhœa* (due to pathogenic organisms such as those of Flexner, Shiga and Y-dysentery).

2. *Dietetic diarrhœa* (due to overfeeding with fat, carbohydrate or protein).

3. *Symptomatic diarrhœa*, that is, secondary to some parenteral infection, such as otitis media, pyelitis, meningitis or bronchitis, which causes an accompanying intestinal upset.

The proportion of each of the above groups among the cases of diarrhœa in summer-time in any country depends on its climate and economic conditions. Thus in America and Australia, where extremes of heat are found, writers regard blood and mucus in the stools as a commonplace, while in the British Isles it is a great rarity.

All cases of summer diarrhœa were at one time regarded as due to an infecting organism, but their subdivision into the three groups to be described must be considered a step forward. In 19 families studied by the author, where there was another child under 2 years of age in addition to the infant patient, in only two families did this other child develop diarrhœa, suggesting that in this group not more than 10 per cent. of the cases are due to a pathogenic organism (infective diarrhœa). An analysis of a group of 84 cases showed that an equal number of the patients had been fed on cow's milk, dried milk, and condensed milk.

Since dried milk is for practical purposes sterile, this is a strong point against the old view that all cases are of the infective type.[1]

A full description of the infective, dietetic and other forms of diarrhœa is given below.

1. **Infective diarrhœa** (epidemic enteritis).—This disease affects infants and very young children during the months of July, August and September, but may be present in sporadic cases throughout the winter months as well. It is characterized by an acute gastro-intestinal upset, with diarrhœa and vomiting, a low blood-pressure, collapse, dehydration, symptoms of intoxication or symptoms resembling shock.

Etiology and pathology.—The organisms most

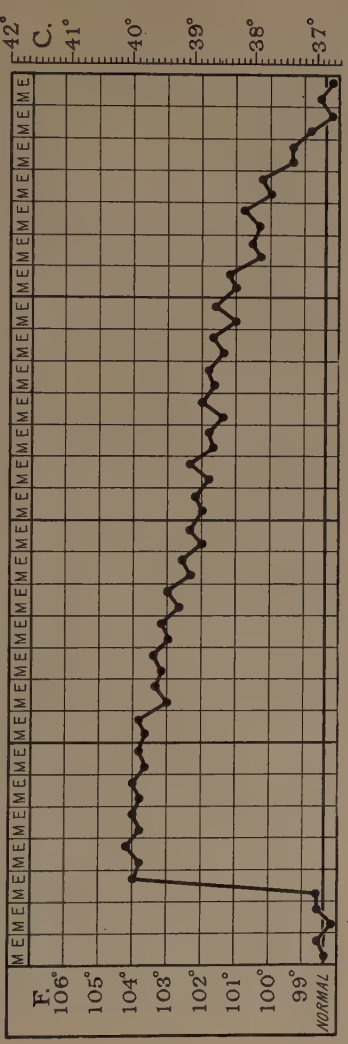

Fig. 18.—Temperature chart of a child with infective diarrhœa.

[1] Donald Paterson and D. N. Nabarro, "Epidemic Diarrhœa," *Lancet*, 1922, ii, 320.

commonly found in the stools are Flexner, Shiga or Y-dysentery, paratyphoid A, B or C, and the Morgan and Sonne bacilli. The last-named is among the most recent additions to the list.

With severe and prolonged diarrhœa, much mucus and frequent blood-tinged stools, one would expect to find gross intestinal ulceration. This is, however, uncommon, although there is occasionally ulceration of the colon and round about the cæcum. The majority of cases show little or nothing in the intestine. The liver is extremely fatty, and almost canary-yellow in colour. How much this is due to the original infection and how much to the treatment, which is often that of starvation, it is difficult to estimate. On section the liver-cells are distended with fat-globules. The kidneys show cloudy swelling, and the glands of the mesentery are in some cases definitely enlarged and congested. A terminal broncho-pneumonia is common.

Clinical picture.—As a rule a perfectly healthy child suddenly develops a high fever (from 102° to 104° (*see* Fig. 18) with acute diarrhœa and vomiting. The stools rapidly become less and less fæcal, and more and more mucus appears. Mixed with the mucus is seen a greater or smaller amount of bright blood. The child from the very beginning is extremely ill. The eyes are sunken, the skin is inelastic, the abdomen becomes scaphoid, the tongue is dry and coated, and the child has the so-called "abdominal" look. Each person who passes the bed is scanned in an effort to attract attention and obtain further drinks, as the child has an unquenchable thirst. Later, if the diarrhœa continues, he becomes pinched and collapsed, and passes into a state of intoxication, the fontanelle being extremely depressed.

Prognosis.—In all cases of true infective diarrhœa the outlook is grave, as there are very few measures which can be undertaken to combat the disease itself. With the general toxæmia from such infections, a large proportion tend to succumb.

Treatment.—If the organism is isolated from the stools or discovered by agglutination, and the appropriate serum is obtainable, it should be used in all cases.[1] The dietetic measures and methods of dealing with dehydration are described under the treatment of dietetic diarrhœa on p. 122.

2. **Dietetic diarrhœa** (overfeeding, fat and carbohydrate dyspepsia).

Etiology and pathology.—In the author's experience the vast majority of cases of diarrhœa in London fall into this group and are due either to—

(*a*) Overfeeding in warm weather on all the food elements, with restriction of the fluids taken in;
(*b*) Overfeeding on fats (cream); or
(*c*) Overfeeding on carbohydrates (sugars and starches).

The infant may be given a suitable feed for cool weather, but with the warm weather the child is less active, there is less heat lost in the breath and from the body, and there is a great loss of fluid through increased perspiration. The infant who was suitably fed for the month of April or May is being grossly overfed on the same amount in July or August. Simple overfeeding in itself is not so bad; it is the continuation of it that brings disaster. If the error be recognized at once and stopped, no real harm is done, but if persevered with, gross permanent damage results.

Dietetic excesses may occur during any part of the year, however, and overfeeding occurs not only in summer-time, especially with reference to some particular food element or elements.

Fat dyspepsia is extremely common all the year round. There are a large number of infants who do not tolerate either creamy milk—even creamy breast-milk—or fatty preparations such as cod-liver oil, or the

[1] Wilburt C. Davison (with Hugh W. Josephs), "The Serotherapy of Bacillary Dysentery in Children," *Journ. Amer. Med. Assoc.*, Vol. 77, pp. 1863-4.

yolk of egg. If such things are persevered with, and more especially in hot weather, the child refuses his food, vomits, passes pale stools and finally has profuse diarrhœa. This is largely due to the formation of fatty acids, which irritate the mucous membrane of the stomach and bowel, with consequent upset.

Carbohydrate dyspepsia, due to overfeeding on sugars or starches, is also common throughout the year, but much more so in the warm summer months. In making alcoholic beverages from starch, if fermentation is not checked the liquid ultimately becomes mainly acetic acid. In a similar fashion sugars and starches offered to an infant in excess pass to the lower portion of the small intestine, where rapid fermentation occurs and the formation of much diacetic acid results. This burns and excoriates the mucous membrane of the bowel, causing increased peristalsis.

Clinical picture.—It is characteristic that the onset of dietetic diarrhœa is not sudden as a rule. If the infant is being weighed daily it will be noticed that he is stationary or even losing a little for several days previous to the attack, and the appetite gradually fails. The temperature slowly rises to 99° or 100° (*see* Fig. 19) with the onset of loose and undigested motions. The infant does not appear acutely ill at this stage. If the feed is persisted with, diarrhœa and vomiting become more severe and the symptoms are aggravated. Dehydration rapidly follows, and the child has sunken eyes, an inelastic skin and parched lips and tongue, with all the symptoms of intoxication. The clinical picture at this stage is very similar to that of an acute infective diarrhœa, but the stools at no time contain blood, although mucus is common and much fæcal material is present. Very little guide as to the offending food element is obtained from the stools once the diarrhœa has started, as quite quickly all the food is rushed through the bowel undigested and mixed with bright-green, unchanged bile.

Where the child is fed on top-cream mixtures, or excessively creamy milk, either fresh or dried, is used,

especially in hot weather, *fat dyspepsia is common*. At first the stools are rather pale or light in colour and liquid. Finally they are mixed with large curds, and other elements of the food come through unchanged. Vomiting is extremely common with fat dyspepsia, as fat leaves the stomach last[1] and tends to remain there and become sour. Cod-liver oil given before a feed is often vomited some three hours afterwards, although the stomach has meanwhile emptied. Pushed to an extreme, fat dyspepsia precipitates an acute acidosis with dehydration.

Where an *excess of starch or sugar* is being given, there is first a tendency to refuse food. The child is often over weight and watery, and ceases to gain,

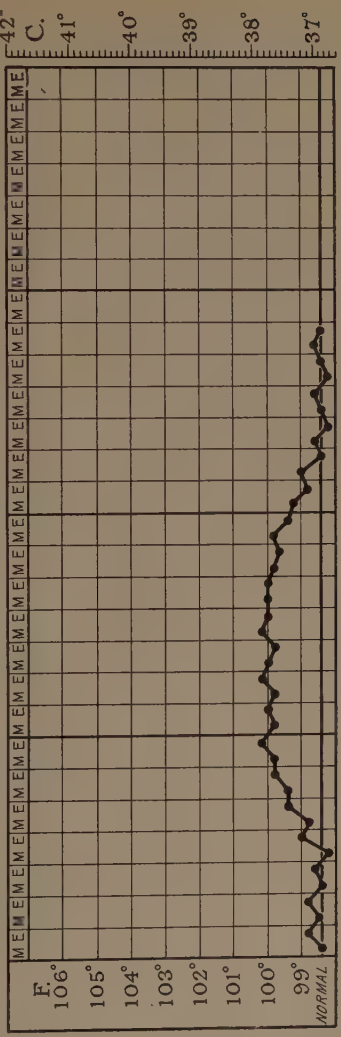

Fig. 19.—Temperature chart of a child with dietetic diarrhœa.

[1] J. Brennemann, "Abt's Pediatrics," Vol. 2, p. 622.

the temperature rising slightly. The motions, which are of a good colour, are rather frequent and slightly acid. If the diet is not modified, the motions gradually become more and more frequent and acid, and burn the buttocks. Vomiting in this type is unusual. Finally the stools contain all the food elements, with many curds and much green unchanged bile. Extreme dehydration follows on the diarrhœa.

Diagnosis.—The clinical pictures of the above three types of dietetic diarrhœa resemble one another closely, and no help is obtained in the final stages of dehydration from an examination of the stools. It is in the early stages, by a careful consideration of the diet and motions, that the dietetic error can be most easily detected.

Prognosis.—In the early stages the prognosis is excellent, but in the later stages, once extreme dehydration has occurred, with fatty change in the liver and much loss of weight, the outlook becomes grave, as the child apparently loses tolerance for all the food elements.

Treatment.—In *slight cases* all food should be stopped for 24 hours, giving a teaspoonful of castor oil first and keeping the child on bland fluids such as plain water, or water to which has been added a teaspoonful of sugar per bottle, or sweetened fruit juice and water. Albumen-water is best not given, as some children tend to have an anaphylaxis to it and show a marked urticaria or œdema with its use. In addition it has no food value. At the end of 24 hours' starvation, *without waiting for the stools to become yellow and smooth and normal in appearance*, the child should be fed. One of the dried milks low in fat-content and high in protein is best given first (*see* Table IX, p. 66); for instance, Half-Cream Glaxo and Dryco are quite suitable, and one of them should be offered at four-hourly intervals. Bland drinks between should also be offered, at hourly intervals, so that the child can quench its thirst and need not rely on its feed for this. *Lactic acid milk*, made according to a modified form of Marriott's method,

is also most useful. B.P. lactic acid, 45 drops, is added drop by drop to a pint of skimmed milk which has been boiled and allowed to cool, stirring the while. Commencing with equal parts of lactic acid milk and water and adding a teaspoonful of sugar to each 5 oz. of the mixture, it should gradually be increased in strength until the child is getting two parts of lactic acid milk to one of water with a teaspoonful of sugar added to each 4 oz. of the mixture. In older infants, say from 5 months of age onwards, a good plan is the addition to the feed, whether a dried milk or lactic acid milk feed, of some starchy preparation such as Savory and Moore's or Benger's Food, on the second or third day after the period of starvation. Eledon, a dried buttermilk made by the Nestlé and Anglo-Swiss Condensed Milk Company, is also extremely useful in such cases.

By far the most alarming aspect of *a severe case* is the dehydration, and this should therefore be treated first. To make up the fluid loss it is best to give **saline** in some form :—

1. By the mouth. If given in half the normal strength there is a tendency for the saline to pass by osmosis through the bowel wall into the body tissues, swelling them. The child will take half-strength normal saline from a bottle in quantities up to a pint per day, with much benefit. Care should be taken that this is discontinued at intervals, thus avoiding a tendency to pulmonary œdema, which results when the treatment is grossly overdone. *Drinks should be offered hourly, day and night.* (Fig. 20.)

2. Subcutaneous saline with 2-per-cent. glucose should be used in those cases where much vomiting is present. Quantities up to a pint in the 24 hours, about 4 oz. at a time, should be given into the thighs, abdominal wall or axillæ.

3. An attempt may be made to give saline rectally with 5-per-cent. glucose, but owing to the straining and tenesmus it is usually returned.

4. Intraperitoneal saline with 2-per-cent. glucose

has not been a great success in the hands of the author. There is a tendency for a dehydrated, debilitated child to become infected very easily, and where the peritoneal cavity is concerned the case may end disastrously.

5. Intravenous saline with 2-per-cent. glucose, or *transfusion* with citrated or whole blood, should be resorted to in extreme cases. Where this can be successfully performed it is very satisfactory.

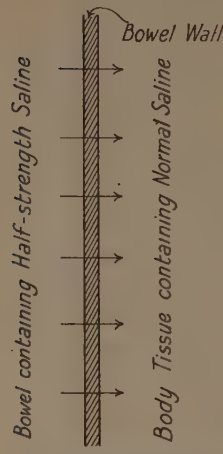

Fig. 20.—Diagram showing rationale of giving half-strength saline to children suffering from diarrhœa.

Frequent drinks of fruit juice and water should be given, sweetened orange or grape juice being added to a bottle of water, or a teaspoonful put in a feeding-bottle of water and offered at hourly intervals both day and night.

Medicinal treatment.—Where the child is collapsed no initial dose of castor oil should be given, but when possible the lower bowel should be gently washed out with warm saline, leaving 3 or 4 ounces in at the end.

Bismuth mixtures may be used such as the Mist. bismuthi co. (Great Ormond Street), the formula of which is as follows:

Bismuth carbonate, 5 grains.
Sodium bicarbonate, 2 grains.
Compound powder of tragacanth, 1½ grains.
Spirit of chloroform, 2 minims.
Caraway water, 1 drachm.

When bismuth is given it is advisable to warn parents that the motions will be darkened by the mixture. *Kaolin* and other similar preparations have been used with some success in the author's hands. *Opium* preparations should on no account be used for infants.

Feeding in severe cases.—This should be along the lines recommended for slight cases, but the increase in the concentration of the feed may require to be a much slower process, and some weeks may have to elapse before the child is having a full feed again.

Stimulants.—Where air-hunger is pronounced, intranasal oxygen should be given. Brandy, 3 to 5 minims, may be given four-hourly, well diluted. Strychnine and adrenalin are useful only in extreme cases.

Unless the infant is extremely ill it should be lifted on to the nurse's knee to be fed and held up afterwards to break its wind, thus preventing the discomfort of wind colic.

There is no doubt in the author's mind that giving sufficient fluid food at suitable intervals is the keynote of the treatment of a severe attack of either dietetic or infective diarrhœa. An example of such treatment is given below :—

An infant weighing 12 lb. with a severe fat dyspepsia is being fed on a lactic-acid milk mixture, with bland fluids between :

	Lactic acid milk mixture taken.	*Sugar water taken.*
1st day . . .	2 oz.	30 oz.
2nd day . . .	6 „	25 „
3rd day . . .	10 „	20 „
4th day . . .	18 „	14 „
5th day . . .	25 „	10 „
7th day . . .	30 „	2 „

If allowed to regulate its own diet in this way the infant will not again be overfed.

In treating a *fat dyspepsia* it is advisable to skim the milk or to use one of the half-cream or skimmed dried milks. Usually patients do well with the feeds thickened with some starchy preparation such as Savory and Moore's or Benger's Food.

In *carbohydrate dyspepsia* (fermentative diarrhœa) a reduction of sugar only is sufficient, with possibly a few hours' starvation. In severe cases it is best to treat along the lines mentioned above, with complete

departure from the high-starch or high-sugar feed the child was having, and the use of lactic acid milk or a half-cream dried milk as a substitute.

3. **Symptomatic diarrhœa** (parenteral infections).—Infections outside the alimentary tract are a much commoner cause of digestive disturbances than is generally supposed. The chief disturbance accompanying acute infections of the ear is often a profuse diarrhœa, and this may even be the case in a breast-fed infant who has been steadily gaining weight. With incision of the drum of the ear and the draining away of the pus the temperature becomes normal and the diarrhœa at once clears up. Too often, however, the attention of the mother and doctor has been diverted from the true site of the infection to the bowel by the presence of diarrhœa, and the real cause is overlooked. Bronchitis and bronchial catarrh, naso-pharyngeal infections, meningococcal meningitis and pyelitis may all be accompanied by acute gastro-enteritis in infants. Mastoid disease may have as its only symptom long-continued diarrhœa which fails to react to dietetic treatment.

Why an infection in some other part of the body should produce a gastro-enteritis is not clearly understood.

Treatment.—A careful examination of the patient should be made in every case of febrile diarrhœa to exclude possible infections outside the alimentary tract. Only in this way will cases of symptomatic diarrhœa be correctly diagnosed, and the treatment should be directed towards the site of infection, not primarily towards the alimentary tract.

Saline solutions.—*Normal saline* is used in cases of severe diarrhœa and shock to replace the fluid lost by the infant, and is made by dissolving a heaped teaspoonful (one drachm) of salt in a pint of water and sterilizing it by boiling. The best method is to inject the saline into the subcutaneous tissues over the chest or abdomen, as it is very difficult to administer it intravenously. The saline is raised to the body-tempera-

ture, and up to 6 or 8 oz. given at a time, depending on the size of the patient. When too much has been given the skin over the site of injection has been known to slough, due to the debility which accompanies dehydration, and therefore great care must be taken not to over-distend the tissues. It is possible to give saline directly into the peritoneal cavity by pinching up a fold of the lax abdominal wall and inserting a needle parallel to the surface, but this method carries with it the risk of infection and of puncture of the underlying gut.

Half-strength normal saline is made up in the same way as the normal saline, but dissolving only a level teaspoonful (½ dr.) in a pint of water. It is of great value given by the mouth, but is not suitable for injection. The child may be given half-strength saline freely as a drink at intervals, or several ounces may be left in the stomach after gastric lavage. It appears to be more readily retained and absorbed from the infant's stomach when given in cases of dehydration. The rationale of this is illustrated by Fig. 20, p. 124.

Saline and glucose.—To combat any accompanying acidosis it is an advantage to give glucose with subcutaneous or rectal saline, and this also acts as a food. With subcutaneous saline the strength given should be from 2 to 5 per cent., and with rectal saline from 5 to 10 per cent.

Olive-oil enema: Olive oil, 2 oz.
Starch, ½ oz.
Water, 20 oz.

Mix the starch into a paste with 3 oz. of water; pour it into the remaining 17 oz. whilst boiling. When cool mix the oil by agitation.

Dehydration Fever

Any infant kept short of fluid for a few hours tends to develop a fever. For instance, in the first three or four days of life, if the colostrum is scanty and the milk slow in appearing, unless frequent drinks of water are

given, especially in summer-time, the child becomes progressively dehydrated and runs a temperature of from 100° to 102°. This subsides at once on the administration of sufficient fluid, and can be prevented by the giving of drinks of boiled water from the age of 12 hours onward, until a sufficient flow of milk has appeared.

Dehydration fever may be seen at all ages. Part of the fever present in acute dietetic diarrhœa is undoubtedly due to the body being deprived of its fluids. Every infant should be offered from 4 to 6 oz. of water daily in addition to its ordinary feeds, and if any considerable proportion of this is taken, the feed should be reconsidered, being probably too small in bulk and calling for further dilution.

Dyspepsias in Older Children

The opinions of various authors to the effect that protein is badly digested and is the cause of numerous evils in infancy and childhood is not borne out by the author's experience. With properly boiled fresh milk or with dried milk the curd (protein) is well tolerated. In regard to older children the tendency among the public is to give too little rather than too much of the various kinds of red and white meat. So rare indeed may protein indigestion be considered that no detailed description need be given of this condition, either in the infant or in the older child.

Fat dyspepsia[1] (chronic acidosis).—Acute fat dyspepsia, giving rise to acidosis or attacks of cyclical vomiting, is described on p. 132, and chronic fat dyspepsia is seen in the same type of child. He is as a rule much under weight, frail and active. Undoubtedly the problem of such a child cannot be solved merely by prescribing a diet. The management of his whole life, and especially of his régime and environment, is of the utmost importance. A congenitally low tolerance for fat cannot be altered any more easily

[1] Donald Paterson, "Gastric Attacks in Childhood," *Brit. Med. Journ.*, May 14, 1927

than the colour of the eyes and hair, but all may become modified with time.

Clinical picture.—Chronic fat dyspepsia undoubtedly accounts for the majority of so-called "thin" children variously described as cases of "nervous exhaustion," "nervous dyspepsia," "debility," "pre-tuberculosis" and "pre-rheumatism." Careful examination yields no definite physical signs of disease beyond infected teeth or tonsils in some cases. More often than not, nothing whatever is found amiss. There is a tendency for the tongue to be continually coated and for bouts of bad breath to occur. The stools are constipated and formed, but spells of pale motions, either formed or loose, are common. At the same time there may be acute vomiting. Examination of the urine shows acetone during these exacerbations, but the presence of acetone may in some cases be detected all the time.

Treatment. *Rest.*—Since these children put so much more of themselves into each waking hour than the normal child, they should have a very short day. A good midday rest, to bed by 6 p.m. and, very often, breakfast in bed is what they need. Under the age of four it is best to keep them off their legs all the morning. After the age of five or six they should be at school, where they sit throughout the morning hours and are only allowed on their legs in the afternoon. School régime suits such children extremely well, and they should be sent to a kindergarten at an earlier age than the normal child.

Diet.—A diet moderately low in fat, but with an increased amount of easily-absorbed carbohydrate is indicated. Details of such a diet are given on p. 134.

Bowels.—The bowels must be kept well opened. Where the child is not under the closest supervision it is best to order medicine as a routine. One of the liquid paraffin emulsion preparations with phenolphthalein is very successful, one to two teaspoonfuls night and morning, with milk of magnesia or Eno's

Fruit Salts given at 7 a.m. on say three mornings in the week. Salvitæ is also of value, but less pleasant to take.

Glucose.—It is the practice of some to give glucose as a routine and undoubtedly in some cases this is quite successful. The following method can be recommended :—

Fig. 21.—Starch dyspepsia. Note the large abdomen. (*British Medical Journal.*)

Take a pound of liquid glucose, add to two pints of water, add orange- and lemon-rind. Simmer until the whole is reduced to a pint and a half, decant and give 1½ oz. of this three times daily, on waking, at 11 a.m., and before going to bed. Clean the teeth afterwards.

The author has not found, however, that such a method is necessary where the mother is intelligent, as extra sugar can be given along with the food or as barley-sugar and the teeth cleaned immediately after, with less risk of dental caries resulting. In any case great care should be taken that the teeth are cleaned after the administration of glucose.

Change of air.—There is no child who benefits to such an extent by frequent change of air as the subject of fat dyspepsia. When small a few weeks three times in the year at the seaside or in some high, stimulating atmosphere is of great benefit. When older it is well that the school should be at the seaside if the home is inland, thus ensuring an absolute change six times in the year.

Starch dyspepsia.[1]—The child with starch dyspepsia differs physically from the fat-dyspeptic chiefly

[1] Donald Paterson, "The Hypotonic (Flabby) Child," *Brit. Med. Journ.*, Feb. 14, 1925.

in the size of the abdomen, which is large, pendulous and tympanitic, being filled continually with wind, and quite out of proportion to the rest of the child's thin body (Fig. 21). The stools tend to be constipated and formed at times, but the constipation alternates with bouts of loose, frothy stools. An examination of the stools will show the presence of much undigested starch, and the iodine test turns the granules blue. Some children suffering from this condition are moderately well-covered, having a large abdomen, but they are extremely flabby and soft, short of breath on exercise and inclined to be anæmic. Such children have been called by Scotch pædiatricians "bread-and-butter" children. The excessively starchy diet formerly in vogue at boarding-schools was apt to produce this type of child.

Starch indigestion is commonest among the poor, starchy foodstuffs being cheap, easily prepared and satisfying.

Treatment.—The essential factor in the treatment of this condition is the reduction of starch in the diet and the substitution of some more easily digested form of carbohydrate. Bread should be given as crisp toast, baked crusts, Ryvita Crispbread, pulled bread or stale wholemeal bread. Porridge, starchy puddings such as rice pudding, and potato should all be reduced or almost abolished. All cereals given should be cooked for at least one to two hours, in order to ensure that the starch granules are all properly split. At the same time the protein intake should be increased. A little more red meat, fish or chicken should be allowed, and at breakfast an increase of crisp bacon or fish. Iron tonics, such as ferri carb. sacch., as much as will lie on a sixpence, three times daily, or Parrish's Chemical Food, one half-teaspoonful three times daily after meals, are specially indicated with such children. Massage and artificial sunlight help to tone up the flabby, soft muscles. For the care of the bowels some liquid paraffin preparation is recommended as a routine.

Cyclical Vomiting (Acute and Chronic Acidosis or Ketosis)

Acidosis or ketosis is characterized by the presence of acetone bodies in the urine and bloodstream and the breath.

Etiology.—To understand acidosis or ketosis it must be considered merely as a symptom with a number of causal factors, rather than as a disease in itself.

1. *The result of infections.*—All febrile illnesses or infections are accompanied by the production of acetone bodies. In ordinary practice the commonest of these infections is tonsillitis.

2. *The result of a badly balanced diet.*—In our present state of knowledge we must look upon carbohydrates as being the fire which burns up the fat, and any deficiency in carbohydrate or excess of fat will leave a certain amount of this latter unburnt, with the result that its breakdown products, the acetone bodies, are formed. The commonest fats in excess in the diet of a child are milk (cream) and eggs.

3. *The result of over-exertion and over-excitement* (physical and nervous exhaustion).—Physical and nervous energy is probably derived from the glycogen stored in the muscles and liver. After an excessive amount of exercise or mental strain, excitement or shock, these stores of glycogen are exhausted and the body-fat is drawn upon, with the result that ketone bodies are produced and the child is in a state of acidosis or ketosis.

In some children there is an inborn tendency towards acidosis, which is undoubtedly hereditary. This hepatic insufficiency can be handed from father to son, and in most of such families one or other parent, it will be noted, suffered either from cyclical vomiting as a child or now has migraine or bus or train sickness.

Pathology.—Nothing is accurately known of the pathology of this condition beyond what has been already stated. If the acidosis is untreated and continues long enough, death may result and the liver

will be found to have undergone an extreme degree of fatty change.

Symptoms and diagnosis.—Cyclical vomiting must be considered an extreme manifestation of the acidosis state. The vast majority of the cases of acidosis are much less well-marked. A typical example of the child who tends to suffer most from acidosis is seen in the subject of cyclical vomiting. As a rule they are boys, thin hypotonic children, who stand badly as if tired, with shoulders drooping and the pelvis pushed forward. They are the over-bright children, often "only" children.

The effects of cardio-vascular instability or asthenia are seen in a tendency to flush easily, or to have pale turns and collapse or faint. The metabolic processes in these children are easily upset, so that they show gastric or liverish attacks with infections, dietetic errors, or over-exertion or excitement. The vomiting may begin acutely, even sips of water being returned, the breath is sighing and they show air-hunger. They become utterly prostrate, and present a most alarming picture, but with proper treatment they recover in a most dramatic fashion from what appears to be a severe illness. They tend to run temperatures with slight provocation, and the tongue is chronically dirty, unless a proper anti-ketogenic (anti-acidosis) diet is adopted.

Treatment.—The treatment should be directed along three lines:

1. *The removal of sources of infection;* bad teeth, septic tonsils, infected ears, or any other source of sepsis must be attended to.

2. *The diet* must be one low in fat, and high in easily assimilated sugars, such as glucose or dextrins. The most common error in the diet of these children is over-feeding on milk (with cream) and eggs. The eggs may have to be excluded altogether, or possibly not more than two in the week may be given. The milk should be well skimmed and reduced to half a pint or less in the day. A good substitute for cow's milk is Horlick's Malted Milk. Apart from this a general diet, such as

that given on p. 93, should be provided. In order to metabolize what fat remains in the diet, however, an excess of sugar should be allowed. Extra sugar on porridge, milk pudding, etc., should be given and stewed fruit should be liberally sugared. At night the juice of an orange, diluted with water and sweetened with four to six lumps of sugar, should be prepared, so that the child may have it immediately on waking in the morning. Sticks of barley-sugar or plain sweets may be given after dinner or tea, and the latter meal should include such sweet foods as honey, treacle, golden syrup, jam, etc. These sweet things should on no account be pressed on the child at any time, but should merely be there should he wish to have them. They should always be offered at the end of the meal rather than at the beginning, so that other articles of diet are not refused because of lack of appetite.

3. *Much more rest* than is usually taken by a child should be insisted upon. These children do best if made to lie down after their midday dinner for half an hour to an hour and sent to bed early at night. Following any excitement or at any signs of being over-tired, they should be put to bed, or kept in bed till after breakfast. The bowels should be kept well open with some alkaline saline. When quite young, say up to the age of four, milk of magnesia should be given occasionally, or, if necessary, as a routine, first thing in the morning, while older children may have Eno's Fruit Salts or Salvitæ or some other saline on waking. Kindergarten is a most useful method of conserving the energies of these children.

TABLE XIII

An Antiketogenic (Anti-Acidosis) Diet

On waking.	A glass of orange, grapefruit or grape juice, sweetened with four lumps of sugar.
Breakfast, say 8 a.m.	Cereals, such as porridge, Groats, Cream of Wheat, Force, Puffed Rice or Shredded Wheat, with a very little skimmed milk and well sweetened with sugar. About two tablespoonfuls of the cereal will

suffice. Second course, two days in the week an egg; three days or four days very thin crisply done rashers of bacon, fried until the fat has been mainly removed and allowed to drip, so that a minimum of fat or grease remains. Fish should be given one or two mornings or lightly cooked liver. Crisp toast and butter, with freshly brewed tea or Horlick's Malted Milk or well-skimmed milk and water, equal parts, one cupful only, complete the meal.

Nothing should be given all morning.

Dinner, say 12.30 p.m. (it is best to have an early dinner for these children).

Fresh mince or stew, liver, underdone beef or steak or cutlet four days in the week, chicken, fish or brains three days; a level tablespoonful or less is sufficient. Potato and green vegetables, a tablespoonful of each, should be given.

Second course, milk pudding or stewed fruit or sponge pudding (not suet pudding).

Water to drink.

Tea, 4.30 to 5 p.m.

Thin bread and butter or crisp toast, with stewed fruit, jam, treacle, honey, golden syrup, or a little milk pudding.

Drinks the same as at breakfast.

In winter a bowl of soup may be given at this meal.

Plain boiled sweets, such as barley-suger, or Mackintosh's toffee, may be given after dinner and tea.

Things to be avoided on account of high fat-content.

The child should not have an excess of eggs or cream, much creamy milk, pastry or suet pudding, chocolate and cocoa.

Constipation in Infancy and Childhood [1]

Constipation in the breast-fed infant.—This is extremely common, and among the causes are:

1. Atonicity of the abdominal walls due to the child being *over weight, soft and flabby*. The condition can be

[1] Donald Paterson, "Constipation and its Management in Infancy and Childhood," *Brit. Med. Journ.*, Feb. 13, 1926.

improved by underfeeding the child and giving massage and sunlight.

2. An *insufficient intake of fluid*, especially during the summer months. In addition to the breast, the child should be offered 4 to 6 oz. of water daily either from a bottle or with a teaspoon from a cup.

3. *Faulty training* on the part of those in charge of the child. The training to pass motions into a chamber should begin in the earliest infancy and be carried out as described on p. 15.

4. If an *anal fissure* is present there is an inclination for the child to fear the act of defæcation. A little ung. hamamelidis should be applied to the anus and the motion kept soft with liquid paraffin emulsion.

5. A certain number of children suffer from *congenital anal stenosis*, that is, the anus is actually too small and requires careful slow dilatation. This is best done by inserting a vaselined little finger on several occasions.

It will be found that a little Semprolin or Petrolagar Emulsion, with or without phenolphthalein, is a great help if given as a routine night and morning. For the child who requires a slight aperient, milk of magnesia (Phillips's) will be found excellent, if given with the first breast-feed in the morning.

6. In the *starved or underfed infant* constipation is often one of the first signs of failure of the milk supply.

Constipation in the artificially-fed infant.—The above remarks are also applicable in great measure to the artificially-fed infant, but in addition some other points must be stressed.

All infants fed on cow's milk are slightly more constipated than the normal breast-fed infant. The higher the proportion of protein or curd in the diet, the more alkaline and constipated the stool. The reverse is true that the more fat and sugar present and the higher the acid content, the more likely is the child to have diarrhœa and sore buttocks. A nice balance must therefore be aimed at between the constipating protein and the acid, diarrhœa-producing fat and sugar.

When an infant is inclined to be constipated and the sugar- and fat-content are not unduly high, a slight increase in this direction is indicated. Mellin's Food, replacing lactose or Demerara sugar, will be found to be distinctly more relaxing. Olive oil, extra cream, and especially castor oil, should be condemned for constipation. For the occasional attack of constipation or formed motions a teaspoonful of Phillips's Milk of Magnesia given first thing in the morning should be sufficient, and for the older infant ½ to 1 grain of grey powder given at bedtime, with the magnesia the following morning, is excellent. As a routine, however, nothing compares with some form of liquid paraffin emulsion such as Semprolin, Angier's, or Petrolagar, and where this is not quite sufficient it should be combined with a little phenolphthalein. In the management of the child it is necessary to attend to the bowel movements with machine-like regularity. Fruit juice, such as orange or grape juice, or prune or fig juice, may be useful as an addition to the diet. In older infants the addition of cereals of the coarse variety such as rusks, crisp toast, wholemeal bread, and such vegetables as sieved spinach, sprouts, greens and cabbage, all help to give the motions bulk and prevent constipation. Where the child is flabby and soft, slight underfeeding, with massage, fresh air and sunshine, tend to firm him up and help to strengthen the abdominal muscles.

The psychological aspect should be considered, even in the infant. He must not be allowed to think that the failure of his bowels to move is of the utmost importance. A studied air of unconcern is to be preferred in the presence of the infant, but the situation should be handled with tact and firmness, making it plain what the child is on the chamber for, and what is expected of him.

Constipation in the older child.—Constipation in the older child has various causes.

1. The mother or nurse may have *failed to establish regular habits* of *evacuation* in infancy. Insufficient

importance has been attached to the emptying of the bowel or insufficient time devoted to this act, with the result that it is put off and neglected, with subsequent constipation. To correct this state of affairs regular habits must be established, the child "sitting down" at the same time daily, and if unsuccessful repeating the process later in the day.

2. Atonicity of the bowel as part of a *general debility* may be found after a debilitating illness or when the child is in bed, due to inactivity. The bowel and abdominal wall become soft and flabby, so that the fæcal contents cannot be pushed forward. This is also seen in flabby soft children, especially those with large, distended abdomens suffering from starch dyspepsia. The "nervous-exhaustion child" or "nervous dyspeptic" described under Fat Dyspepsia (*see* p. 128) also falls into this class. The muscles are hypotonic and very little intra-abdominal pressure can be exerted on the intestinal contents.

Until the general condition of such children is improved, no improvement in muscle-tone is likely to occur. This may mean changing the child's whole life and régime. A change to the seaside, general tonics, fresh air, real or artificial sunlight, massage, adjustment of the diet, are all indicated.

3. *Anal fissure* or *hæmorrhoids* do occur in older children, although uncommon. In all cases where pain is complained of, a careful examination should be made to exclude these, and galls-and-opium ointment or ung. hamamelidis (witch-hazel ointment) should be applied both before and after defæcation.

4. The normal child who is getting *insufficient food* or food which is too completely absorbed will tend to be constipated. The diet should contain a certain amount of residue-producing food. Such is to be found in wholemeal bread and cereals of all kinds, and especially in vegetables and fruit, and a liberal supply of each of the last two should be present in every child's diet. It is seldom that green vegetables or raw or cooked fruit are given too freely, the great tendency

being to underfeed children on these articles of diet. Bemax is also useful in this respect.

On the other hand the diet may be liberal, but *wrongly balanced.* A diet containing too much protein will undoubtedly tend to produce constipation, as protein gives rise to alkaline motions which decrease peristalsis.

Careful review of the child's food with these points in mind will often reveal the error.

5. The diet of many children is *deficient in fluids,* and especially may this be said of children in summer-time. In this way the motions are allowed to become dry and crumbly. Drinks of water should be given on waking and during warm weather, even between meals, and every child should drink from one pint to a pint and a half of fluid daily as a minimum. Quantities less than this will tend to produce a dry intestinal content.

6. In the older child at school it is essential that *sufficient time* be given for proper attention to the bowels. No day should elapse without a motion, and the child should be trained to report his failure at once, so that the proper steps can be taken to ensure one the following morning.

Medicinal treatment.—In older children *liquid paraffin emulsion* or even plain liquid paraffin should be relied upon. Such preparations as Petrolagar, Semprolin and Agarol, especially when combined with phenolphthalein, are most efficient. Two teaspoonfuls night and morning as a routine, with occasionally some Eno's Fruit Salts or syrup of senna last thing at night or first thing in the morning in addition, should keep the most obstinate bowel in order. *Castor oil* should be avoided in all cases of constipation, as it tends to leave the child more constipated than before, and should be reserved for those children who are inclined to have diarrhœa. *Senna-pods* may be given daily or small doses of *cascara,* as a bowel tonic. *Taxol,* one to two tablets daily, will be found extremely satisfactory. Vegetable laxative tablets, prepared by various firms, are useful as an occasional aperient.

Salvitæ and other forms of saline are best given at least an hour before breakfast, in order to ensure an efficient result.

Some old Great Ormond Street favourites are:

Mist. Rhei cum Soda

Rhubarb in powder, 1 grain
Sodium bicarbonate, 2 grains
Syrup of ginger, 6 minims
Peppermint-water to 1 drachm.

Mist. Aloes Co.

Tincture nux vomica, 1 minim
Tincture of ginger, 2 minims
Extract of hyoscyamus, $\frac{1}{3}$ grain
Aloes, $\frac{1}{10}$ grain
Syrup of senna, 15 minims
Dill-water to 1 drachm.

Mist. Cascaræ

Liquid extract of cascara
Liquid extract of liquorice
Syrup of orange-peel
Chloroform-water
} of each 15 minims.

Mist. Cascaræ cum Malto

Liquid extract of cascara, 10 minims
Extract of malt to 1 drachm.

Syrup Ficorum Laxans

Fluid extract of senna-pods, 6 minims
Fluid extract of cascara, 3 minims
Compound tincture of rhubarb, 3 minims
Syrup of figs to 1 drachm.

Rumination (Merycism)[1]

This is the term used to describe a habit which some children develop of vomiting or spitting up their food, and re-swallowing a portion of it.

Etiology.—Very often the food is a pleasant one, such as one of the sweetened condensed milks, and it has been given in too dilute a form, so that the total

[1] Donald Paterson, "Rumination (Merycism) in Infants," *Practitioner*, Dec. 1928.

feed is greater than the stomach can contain. The result is that natural vomiting occurs at first, and the infant soon acquires the trick of being able to regurgitate its food at will. It has been said that some acute digestive disturbance may initiate the vomiting and that it continues afterwards as a habit, but this has not been the author's experience. It is claimed also that rumination is a family disease, which can be traced through the generations. This also cannot be borne out by the author.

Clinical picture.—As a rule the child presents itself very much under weight, thus at say six months it may weigh only 7 or 8 lb., which is very little more than birth weight. The story is that from a very early age, a few weeks only, the infant brought its food up in small amounts almost continuously from one feed to another. The pillow was always wet with its vomit. In many cases the regurgitation can hardly be called vomiting, but rather spitting. On close inspection the infant can be seen to hollow the tongue like a funnel, champing the jaws and struggling as if uncomfortable. Occasionally the back is arched. Suddenly the food wells up in its mouth, a small portion spilling from the corner and the remainder being slowly re-swallowed. This process is repeated over and over again until a considerable proportion of the food is wasted.

Diagnosis.—It is seldom that the clinical picture which has been described is seen by the physician or even the mother, as the infant, usually a girl, bright and clever, will not ruminate while anyone is present. The process is best studied by placing a screen round the head of the bed and peering over the top. The infant, when she thinks herself alone, will go through the process described.

Treatment.—There can be little doubt that rumination is a purely functional complaint. The spitting and vomiting will cease in the following circumstances :

1. If from one feed to the next the child is watched and interested, held up to look out of the window, played with and talked to, no return of the food occurs.

2. If by some mechanical means the jaws are prevented from opening and champing a cure occurs. A small skull-cap with broad strings passing from the region of the ears under the chin and fastened on the top of the head again, thus firmly clenching the jaws, will act as a deterrent. Unless the mouth can be easily opened and closed the infant fails to get the pleasure from ruminating which makes it worth while.

3. If the food is thickened with some starchy preparation, so that it is small in bulk and firm in consistency, ruminations cease. Such starchy substances may be Benger's or Savory and Moore's Food, Robinson's Patent Groats, Cream of Wheat, semolina, Farina or Wheatena. The author has found Benger's Food of great service in such cases. It is well always to give cod-liver oil in some form at the same time as the thickened feeds, as the child as a rule makes such extremely rapid progress that it tends to develop acute rickets unless this precaution is taken.

4. If during the process of rumination a drop of quinine solution is placed in the open mouth the face of the child shows its disgust and for the time-being all efforts at rumination cease.

Prognosis.—On thickened feeds children given to rumination gain weight rapidly, and the mortality is extremely low.

Aerophagia (Wind-swallowing)

The swallowing of wind is a normal physiological occurrence in every infant, breast- or bottle-fed. An X-ray of a normal infant will convince an observer of this fact, as a bubble of air gathers at the fundus of the stomach (Plate 3). Brennemann has shown that the thicker the feed the less wind swallowed with it.

Every infant should therefore be held up for from 15 to 20 minutes after each feed, until the wind is broken or eructated twice, if undisturbed sleep is to follow. Unless properly held up the air is either eructated while the child is in a recumbent position, in which case part

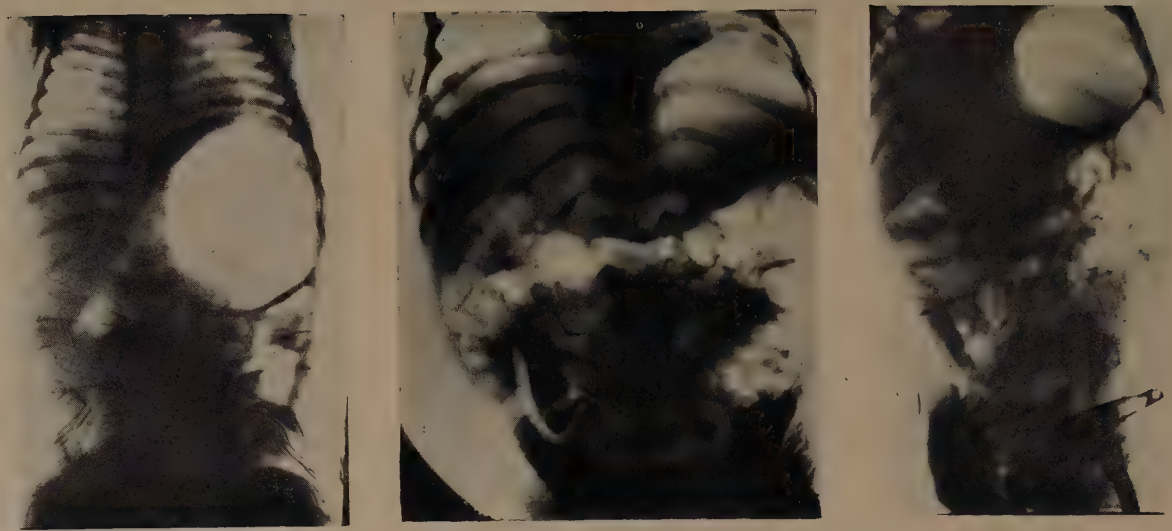

Plate 3.—Radiograms of normal infants immediately after a bottle-feed. Note the wind in the stomach of each infant and its presence in the large intestine from previous feeds.

of the feed is brought up at the same time and wasted, or the air passes through the pylorus into the bowel, causing colicky pains, distension and increased peristalsis.

Slight degrees of wind-swallowing are physiological, but the habit may become pathological very easily. An extreme example of this is seen in infants who are grossly underfed on the breast or bottle, or in infants who lack the inborn ability to suck properly, mouthing the teat rather than fastening on to it with a tight suction.

One of the commonest mistakes made is to give the feed through a teat having too small a hole. It is commonly taught to mothers and nurses that wind is swallowed because of too fast a teat, but this is a fallacy. At each suction a small quantity of air is swallowed, and if say 500 suctions are required for the infant to take the feed, more wind must necessarily be swallowed than for say 300 suctions with a larger hole. With a very fast teat gulping and spluttering result, but the hole should be of a sufficient size to allow the infant to get the whole feed in from 7 to 10 minutes. Then from 10 to 15 or even 20 minutes should be spent in holding the infant up.

Treatment of wind-swallowing.—1. Offer the infant a sufficient supply of food for his needs. If breast-fed, make sure that he is not being underfed.

2. See that he gets his food easily. Enlarge the hole in the teat if necessary and do not leave him at the breast longer than is necessary for it to be thoroughly emptied. This can be judged by doing test-feeds.

3. Hold the infant up for sufficient time after each feed for him to break his wind twice.

4. For a week or two after commencing treatment give the infant (three months of age) a grain of chloral three or four times daily, so that he approaches the feed in a less frantic condition.

CHAPTER V

DISEASES OF THE EYE, EAR, NOSE AND THROAT

Diseases of the Eye

Squint in childhood.—Organic squint may be due to some organic brain lesion following an infection, cerebral hæmorrhage or birth injury, or to paralysis caused by maldevelopment of the sixth nerve.

Functional squint (convergent or divergent concomitant squint).—This is the usual squint found in infancy and early childhood. The child appears to look at things with one eye and squint with the other.

Etiology.—The cause as a rule is some refractive error, and this may be accentuated by any debilitating illness, such as measles or whooping-cough.

Treatment.—The treatment should be commenced at an early age, to prevent the loss of sight in the squinting eye, as the child tends to see with the good eye only. By the instillation of atropine and bandaging the good eye, the defective or squinting eye is forced into use.

Conjunctivitis.—Various forms of conjunctivitis common to adults are also seen in childhood. That due to the Koch-Weeks bacillus is probably the commonest. Bathing the eyes with boracic lotion or lotio hydrarg. perchlor. 1 in 10,000, or instilling drops of silver nitrate 1 per cent. or Protargol 2 to 5 per cent., are considered the best treatments. Zinc sulphate 1 per cent. acts efficiently in this infection.

Ophthalmia neonatorum has already been described on p. 39, and interstitial keratitis is referred to on p. 484.

Phlyctenular conjunctivitis.—This condition is most commonly found in the so-called "strumous" child.

Etiology.—At one time it was thought to be a manifestation of tuberculosis, but it is now considered to be a sign of great debility only, and such children are good soil for tuberculosis. A diet deficient in fat-soluble vitamins, confinement indoors, lack of sunshine and good food, are often contributing factors, as also are septic teeth and tonsils.

Clinical picture.—Small whitish or yellowish nodules are seen, situated on the conjunctiva, surrounded by an area of radiating, engorged venules. In some cases photophobia is marked and the child complains of much discomfort. Actual ulceration of the cornea may occur in some cases, leaving opaque nebulæ.

Treatment.—The application locally of atropine ointment (1 per cent.) is advisable. General treatment, however, is most necessary. Septic teeth and tonsils should be removed, cod-liver oil and malt should be given, and artificial sunshine will be found of benefit. A change to the seaside also helps recovery, but there is a marked tendency to recurrence.

Chronic blepharitis.—This is often present in the strumous or "exudative diathesis" child, the child who is plump, watery, flabby and who often has an extremely good colour. All the mucous membranes are easily infected by catarrhs in these children, and blepharitis is common. Bathing with 2-per-cent. sodium bicarbonate lotion, followed by the application of yellow ointment (ung. hydrarg. ox. flav.) is most beneficial. An eye surgeon should be consulted in case there should be some refractive error.

Styes.—These are due to an infection of the Meibomian glands, resulting in the formation of minute abscesses. Hot bathing and pulling out of the appropriate eyelash are most needed. Occasionally the abscess may require to be incised. Where styes tend to recur a course of injections of staphylococcal vaccine is most helpful, as it is also in chronic blepharitis.

Proptosis or exophthalmos may be found in various conditions in children. *Exophthalmic goitre* is extremely rare before the age of 12 to 14. In *scurvy* there may be hæmorrhages round about and behind the eyeball, actually causing protrusion. With a *new growth*, such as Hutchison's type of renal sarcoma, there is a marked tendency to proptosis, due to cerebral metastasis. With *inflammations of the wall of the orbit*, especially on the nasal side, an extra-orbital inflammation with an abscess develops. This may extend from inflamed turbinate bones and really constitute an osteomyelitis. In some cases seen by the author it was found necessary to drain the abscess by incision and resection of the periosteum of the orbital bone, an operation which was completely successful.

Diseases of the Ear [1]

Otitis media. **Etiology.**—Earache in the child is most commonly due to an inflammation, catarrhal or otherwise, of the tympanic membrane or surrounding parts. The inflammation, however, may be in the Eustachian tube, causing an obstruction of the passage of air to the middle-ear and bulging or retraction of the drum. Foreign bodies in the external auditory meatus, such as wax, peas and beads, may also give rise to earache, and earache is quite common after the operation for the removal of tonsils and adenoids.

Epidemic infections with certain organisms seem to have a predilection for the ear. For instance, in the early part of 1929 an epidemic of the influenzal infection then prevailing tended to produce otitis media in an undue proportion of cases among small children.

Otitis media is a common complication of measles, scarlet fever and diphtheria.

Clinical picture.—Preceding otitis media there is as a rule a catarrhal infection of the nasopharynx. The child has a cough and there is a discharge from the nose, generally with some fever. The infection spreads

[1] T. T. Higgins, in Garrod, Batten, Thursfield and Paterson's "Diseases of Children" (Edward Arnold & Co., 1929), p. 304.

from the Eustachian tube to the middle ear, the temperature rises rapidly and intense pain is felt in the ear. The infection is usually bilateral.

On examination the drum of the ear is red and has lost its lustre, bulging being present. If no treatment is instituted, in 24 to 48 hours, during which time the pain waxes and wanes, the drum will burst, a purulent discharge taking place. The streptococcus, pneumococcus, micrococcus catarrhalis and influenza bacillus may all be found in this discharge, which continues as a rule for days, weeks or even months, gradually subsiding. Accompanying the infection of the middle ear, there is often an infection of the mastoid process.

In the infant with otitis media, suspicion may not fall on the ears merely from observing the child. An examination of the drums should be made in every case. The child often exhibits a degree of toxæmia and may even behave as in meningitis, holding the head back and screaming at intervals. Most frequently there is an accompanying tonsillitis, when the glands in the neck are enlarged. The baby's action in putting its hand to its ear cannot be taken as a sign of earache, as a normal infant will often do the same thing.

Prognosis and treatment.—Earache in children should always be taken seriously, and a careful examination of the drums made. If wax is present, the ear should be syringed out with a Higginson's syringe, using *tepid alkaline lotion*, e.g. a teaspoonful of sodium bicarbonate in a tumblerful of lukewarm water. If the drums are red and early bulging is detected, paracentesis should be undertaken. A golden rule for the practitioner is that when in doubt paracentesis should be performed. A thin sero-purulent discharge will then take place for the next two or three days to a week, which later becomes scanty and thick, finally drying up and the tympanic membrane healing. If the drum is allowed to burst a small portion is destroyed and complete healing does not so commonly take place, the discharge continuing very often for some weeks. Fomentations to the ears or a hot bottle under the head

are most valuable if such treatment is instituted early, when the earache first commences, and continued until the discharge has ceased. With very slight earache, before redness can be detected, drops of carbolic and glycerin should be instilled:

Carbolic acid 2½% in glycerin } of each
Distilled water } equal parts.

Great care should be taken that the external auditory meatus is not allowed to become infected by the discharge from the ears, as an eczema may develop giving rise to a chronic discharge. To prevent this, drops of rectified spirits are useful:

Boric acid, 8 grains.
Alcohol, 2 drachms.
Water, 2 drachms.

The directions for treatment at the Hospital for Sick Children are somewhat along these lines. First the mother is directed to procure a glass syringe from the chemist and to make up a suitable quantity of the boracic or the alkaline lotion, bringing it to a tepid temperature. Next she is to syringe the ear gently with the mixture until it is clean, then thoroughly dry with a piece of cotton-wool, making sure that all the lotion is dried from the ear. Then she is to make a spill from paper and blow a small quantity of the powder into the ear (the powder consisting of iodoform powder, one part; boracic powder, three parts). This should be done twice daily.

Acute mastoiditis. Etiology.—This condition as a rule accompanies acute otitis media. In the infant its onset may be very rapid, and within 24 hours the clinical picture may be complete. The infection usually spreads from the middle ear to the mastoid antrum, infecting the mastoid cells. The organisms are the same as in acute otitis media.

Clinical picture.—The child becomes drowsy and lethargic. The temperature is high, and vomiting may take place, accompanied by headache. Very often with the onset of mastoiditis the earache ceases and

the child may appear quite bright at intervals, although generally drowsy. On careful examination there may be a slight œdema over the mastoid process, or the œdema may be marked, with displacement forward of the ear. Occasionally, however, no œdema is present and there is little or no tenderness over the mastoid. Redness and swelling of the posterior wall of the external auditory meatus is a help in such cases, and trans-illumination, showing opacities in this region, is also of value.

Treatment.—The treatment is surgical and should be considered an emergency operation, the mastoid being opened immediately and freely drained.

Complications.—Infection of the lateral sinus and meningitis are the two most common complications, but occasionally cerebral abscess supervenes.

General remarks: Prevention of otitis media and mastoid disease.—Undoubtedly early removal of infected tonsils and adenoids is by far the best mode of prophylaxis. Certainly this should be done in every case in which there has been earache. Septic teeth should also have attention.

Ear discharge (otorrhœa).—The discharge may be from the external auditory meatus, due to the presence of some foreign body, or to a chronic eczema, or from the middle-ear, due to an otitis media. The ear should be carefully syringed out with a Higginson's syringe, using a silver Eustachian catheter or special glass nozzle and an alkaline lotion. The removal of septic tonsils and adenoids should be undertaken in every case of chronic ear discharge due to middle-ear infection, as this is often the best and only means of doing away with the infection (*see* Otitis media, p. 147).

Infections of the Nose and Throat [1, 2]

Infections of the nose and throat, or upper respiratory tract infections are extremely common in childhood.

[1] Joseph Brennemann, "Throat Infections in Children," *Arch. of Ped.*, Vol. 42, March 1925, pp. 145-168.

[2] Donald Paterson and G. W. Bray, "Tonsillar Hypertrophy and Infection as a Factor in Ill-Health in Childhood," *Lancet*, Nov. 24, 1928, p. 1074.

Nothing plays so large a part in producing the minor ailments of infancy and childhood.

Etiology.—The infecting organisms may be the micrococcus catarrhalis, the pneumococcus, streptococcus or influenzal bacillus. It is claimed by some that the infecting organism of the common cold is ultramicroscopic or a filter-passing virus. These infections are easily passed from one member of a household to another, and there can be little doubt that sore throats and infected adenoids with nasal discharge are commonly contracted from some other child. They may, however, be brought about by sudden change from a warm, stuffy atmosphere to a cool atmosphere when the normal organisms of the nose and throat may obtain a hold on the chilled-off mucous membrane. Accompanying measles and scarlet fever there is usually well-marked infection of the nose and throat, and this may occupy such prominence in the picture that the rash is almost overlooked.

Pathology.—The inflamed lymphoid tissue of both adenoids and tonsils drains into the glands of the neck, and the tonsillar glands at the angle of the jaw on each side become enlarged where the throat is septic. Where the glands have been previously infected repeatedly there is a tendency for the organisms to spread further down the chain of glands or actually to reach the blood-stream. Following on nose and throat infections due to the organisms spreading, various other organs in the body are affected. These will be dealt with under Complications.

Clinical picture and symptoms.—The severity of nasal and throat infections varies enormously. It may be merely a slight running nose on the one hand, or it may develop into a severe quinsy or Vincent's angina on the other hand. The temperature may be slight or high. If the mucous membrane of the nose and the adenoids are involved there tends to be a headache. When the tonsils and pharynx are involved there may be pain on swallowing and general fever, lassitude and drowsiness. The whole infection may settle down in

24 hours or continue for weeks or months, depending on the organism and the child infected. Coincident with each infection the adenoids may swell, causing nasal obstruction and snoring. Between attacks this may not completely subside, so that broken sleep and restlessness are a feature.

Complications.—The complications of upper respiratory infections are many and varied and are of the utmost importance. *Laryngitis* and *bronchitis* commonly follow on catarrh of the nose or throat. *Otitis media* is the direct result of the same thing. *Umbilical colic* is often associated with sore throats. It is difficult to explain the etiology of this, but it is probably due to enlarged mesenteric glands, caused either by swallowed infection which has passed through the intestinal wall to the glands, or by infection reaching the abdominal glands by way of the bloodstream. It is much more likely to be the former.

Acidosis attacks.—Accompanying an acute sore throat or catarrh of the nose the child may have acute vomiting, probably due to a sudden formation of acetone secondary to the infection. This may be accompanied by constipation or, on the other hand, by diarrhœa. If the infections appear over and over again at regular intervals a wrong diagnosis of cyclical vomiting may be made.

Rheumatism.—The infection of rheumatism almost certainly gains entrance to the body by way of the tonsils.

Nephritis.—The acute hæmorrhagic type of nephritis is most commonly the result of septic tonsils.

Stomatitis.—This may also result from infections of the tonsils, especially with the spirochæte of Vincent and the fusiform bacillus.

Peritonsillar abscess.—This is uncommon before the age of five or six.

Retropharyngeal abscess.—This is most common in the first few months of life. Both these types of abscess are sequels to throat infections.

Upper respiratory infections undoubtedly precipitate *asthmatic attacks* in a great many cases.

In older children *disease of the antrum* may be directly traced to an infection from tonsils and adenoids.

Treatment.—In all cases the child is best in bed, as there he is less likely to become chilled and can less easily convey his infection to others. The temperature of the room should be about 60° F., and the air is best kept slightly moist with occasional small amounts of steam containing friars' balsam. Fifteen minutes every three or four hours is sufficient. Spraying the inflamed surface with liquid paraffin containing a drop or two of oil of eucalyptus to the ounce is to be recommended. A mixture of potassium chlorate and sodium salicylate every four hours is most beneficial. For a child of three the mixture should be:

Pot. chlor., 2 grains.
Sod. sal., 3 grains.

Gargling is practically useless in childhood, as it cannot be accomplished efficiently. Painting the throat is distressing to the patient, but where a follicular tonsillitis is present or the tonsils are much swollen one good painting with a weak tincture of iodine is often most effective. The bowels should be kept well open with grey powder at bed-time and milk of magnesia or some saline such as Eno's Fruit Salt first thing in the morning. The diet should be that for a fever patient (*see* p. 415).

Operative treatment.—There is no more successful operation from the clinician's point of view than the removal of septic tonsils and adenoids in childhood. Children who have been ailing and pale, with a tendency to earache or actually discharging ears, with repeated feverish turns and gastric attacks, become surprisingly well after tonsillectomy. The operation for choice is that of tonsillar dissection, as taught and practised at Great Ormond Street. In this way the lingual portion of the tonsil is obtained as well as the fossule portion. Guillotining the tonsils, except in the hands of a great expert, may not be entirely successful, as the lingual portion is often left behind, though it has the advantage

of a short anæsthetic. The tonsils should not be removed before the age of three unless there is some urgent complication, such as discharging ears, and it is best if they can be left until the child is four or five. If, however, they are definitely enlarged and give evidence of being septic by enlargement of the tonsillar glands, they are best removed as soon as possible, as much for prophylaxis as for treatment.

Injectio Potassii Bromidii Composita

(For rectal injection after tonsillectomy).

	Age, 1 *year.*	*Age,* 2 *years.*	*Age,* 4 *years.*
Potassium bromide .	10 gr.	20 gr.	25 gr.
Aspirin . . .	5 gr.	10 gr.	15 gr.
Mucilage of tragacanth . . .	½ oz.	½ oz.	6 dr.
Normal saline solution to . .	2½ oz.	3 oz.	5 oz.

Anæsthesia.[1]—Although paraldehyde by rectum has only recently been tried as a pre-medication before anæsthesia it has great promise. The child goes to sleep some time before the operation and awakes some hours later with no knowledge of the operation whatever. A drachm of paraldehyde is administered rectally for each 14lb. of the child's body-weight about an hour before the operation. It is best given dissolved in saline and 5 per cent. glucose.

The function of the tonsil.—Opponents of the removal of tonsils by operation ask—If tonsils are given us for some reason, and if it is true that they act as a filter and prevent infection passing from the outside of the body into the bloodstream, why should they be removed? From early infancy to the age of three, four or five, children are continually getting slight tonsillar infections. On each occasion there is a mild engorgement and enlargement of the tonsil and antibodies are formed against various types of organisms, conferring some degree of immunity on the individual. It is a slow process of vaccination against myriads of organisms and seems necessary to protect us in later life. The removal

[1] H. Sington, "Pre-medication by Paraldehyde in Children," *Proc. Roy. Soc. Med.*, 1929, Vol. 22 (Sect. Anæs.), p. 7.

of tonsils, then, at too early an age would to some extent prevent this process taking place, but once it has taken place the tonsils seem to have served their purpose, since they become definitely infected themselves and act as a source of sepsis rather than an aid against it. Tonsils which have been removed show on section tuberculous infection in some cases, while in other cases they are actually bags of pus. This may occur at almost any age, but is extremely common between the ages of three and five, when indications for the removal of tonsils may be looked for.

Glandular Fever (Infective Mononucleosis)

Etiology.—The causation is unknown. Children between the ages of five and eight are most commonly affected. There seems little doubt that this is an acute infectious disease, readily transmissible from one child to another, and epidemics are seen, especially in institutions.

Clinical picture and diagnosis.—The incubation period is from 7 to 10 days. The child becomes acutely feverish, and the glands in the neck, especially those beneath the sternomastoid, become swollen and tender. At the same time there may be vomiting and abdominal pain, and a general glandular enlargement, in the axillary and inguinal regions especially. The blood-count shows a leucocytosis with a relative increase of the lymphocytes up to 60 per cent. The liver and spleen are enlarged and palpable. Due to the enlarged cervical glands the neck is stiff and tender, and there is pain on swallowing. There may be very little tonsillar involvement. Occasionally hæmaturia occurs. Convalescence is prolonged.

Treatment.—The head should be splinted (*see* Fig. 71, p. 434). A mixture of pot. chlor., grains 3, and soda sal., grains 5, should be given four-hourly. Great attention should be paid to the cleanliness of the mouth. During convalescence iron tonics and a change to the seaside are indicated.

CHAPTER VI

DISEASES OF THE DIGESTIVE SYSTEM

Stomatitis

The simplest form of stomatitis is "*thrush*," due to Oïdium albicans.

Etiology.—Thrush is an infection of the mucous membrane and submucous tissues by a vegetable fungus, the spores of which project from the surface and give the characteristic white appearance. It is commonest in infants who are debilitated and who become infected from contaminated dummies and teats.

Clinical picture.—The tongue, roof of the mouth and inside of the cheeks have a white, patchy or furred appearance, as if the infant had just been drinking creamy milk and thin clots had been left behind sticking to the mucous membrane. An attempt to wipe these off, however, shows the patch to be firmly adherent to the tongue or cheek. There is a tendency for the infection to "work through" the child, so that it is accompanied by severe vomiting and diarrhœa.

Treatment.—Glycerin and borax, or a simple mixture of chlorate of potash, water and glycerin, applied to the mouth will clear up the condition in a day or two. It is most efficacious also to wrap a piece of muslin about the finger and gently scrape the affected part until the patch is more or less removed. When the spores are broken off in this manner they fail to reappear.

Thrush may also be treated by freshly prepared 1-per-cent. aqueous solution of gentian violet, applied to the mouth twice daily for three days and once every day for two weeks.[1]

[1] H. K. Faber and Esther B. Clark, "Prevention and Treatment of Thrush," *Am. Journ. Dis. Child.*, Sept. 1927, p. 408.

APHTHOUS STOMATITIS

In this form of stomatitis small white patches appear on the tongue, cheeks and gums. The superficial layer of mucous membrane becomes necrosed, due to some mild infection, with the result that it later sloughs off, leaving a small red, raw area or ulcer. It is the author's opinion that there is no sharp line between the various forms of stomatitis described, but merely a difference of degree.

Etiology.—Whether the aphthous stomatitis be due to a specific organism or to the organisms of the mouth gaining a foothold because of the debilitated condition of the patient, or to a lowered antiseptic power of the saliva, is not known. Certainly aphthous stomatitis is most often seen when the health of the patient has not been good for some time. It is not commonly found in infants, the older child being most often affected.

Treatment.—As for ulcerative stomatitis, set out below.

ULCERATIVE STOMATITIS

This infection has been described under the various heads of " trench mouth," " Vincent's angina," etc. It is impossible to say whether it is the lowered resistance of the patient or the virulence of the infection which is the outstanding factor in the clinical picture.

Etiology and pathology.—The organism in this form is in most cases the spirochæte of Vincent and the fusiform bacillus. The condition is extremely infectious and tends to affect Poor Law institutions, asylums and schools, or groups of men in barracks. In families the mother or father may be the source of infection, having chronic pyorrhœa. In private practice it is often the nanny who is at fault. Why the infection should affect the tonsils only at one time, as an epidemic of ulcerative tonsillitis, and the mouth only at another time, as an epidemic of ulcerative stomatitis, cannot readily be explained.

Clinical picture.—Some cases are extremely mild, there being redness of the gums with swelling and œdema

only. This causes the teeth to appear to sink back into the gums, just the tips being visible. Ulcers appear at the base of the teeth in other cases, and the portion of the cheek coming in contact with these ulcers also becomes infected. There may be sloughs or ulcers on the tongue. All degrees of severity, from small aphthæ on the one hand to large gangrenous areas described as "noma" on the other hand, may be caused by this infection.

Temperature.—The onset is quite sudden, the temperature rising in a few hours to 100° or 101°, the child becomes fretful, refuses its food and tends to dribble from the mouth. In a certain proportion of cases the tonsils are affected and in severe cases the saliva is fœtid. The infected small infant is extremely ill. Profuse vomiting and diarrhœa may occur before the mouth can become seriously involved. Teething infants show the severest lesions, possibly because of the lowered vitality of the gums when the infection takes place.

Treatment.—In the mild form chlorate of potash, 2 grains four-hourly, may be sufficient. Glycerin and borax or glycerin and perchloride of iron applied hourly to the gums may also clear up the infection. There is little doubt in the author's mind, however, that Bowman's mixture, which is as follows, is the most efficient method of treating this group of stomatitis:

Liquor arsenicalis, 2 drachms.
Vinum ipecacuanhæ, 2 drachms.
Glycerin, 2 drachms,
Aq. menth. pip. ad 1 oz.

For children of 2½ to 5 years of age, who will spit out the medicine, it can be applied directly to the gums and tongue by means of a soft camel-hair brush and any excess expectorated. For those children who are too young to run this risk, however, 5 drops should be added to a drachm of water. This should be applied to the tongue and gums 4 or 5 times daily by means of a piece of muslin wrapped round the little finger and dipped in the mixture. The action of the arsenic is to kill the spirochæte and that of the ipecacuanha to kill the

fusiform bacillus. The glycerin makes the mixture adhere to the affected parts, and, since it is hygroscopic, it promotes a free flow of lymph. Other antiseptics have a reputation, and undoubtedly succeed, but probably none has such rapid and sure success as the mixture recommended.

Care should be taken to see that the bowels are well open, and for this purpose some mercury preparation such as grey powder, with magnesia in the morning, is most suitable. The diet should be an anti-ketogenic one (*see* p. 134).

Cancrum Oris

This is a condition rarely seen at the present day. It commences as a small patch or ulcer on the inside of the cheek in children who are debilitated for any reason. This ulcer spreads rapidly and may actually perforate the cheek.

Pathology.—It is probable that the causal organism is in most cases the spirochæte of Vincent and the fusiform bacillus, acting in a child who is extremely debilitated. Such a condition sometimes occurs in blood diseases or long-continued chronic illnesses.

General treatment.—Since one of the chief factors is the general condition of the patient, any treatment must be directed towards raising the child's immunity and his ability to throw off the disease. Treatment of the underlying condition, then, is most important. Tonics, change of air and good dieting are essential, and artificial sunshine is sometimes a help.

Local treatment.—The application of antiseptics such as strong carbolic to the actual site of the lesion is advisable, and iodine, chloramine and Eusol are also most useful as mouth-washes, to irrigate the ulcer and as local applications over the wound if perforation has already taken place. Probably the most efficient mouth-wash or application is the arsenic and ipecacuanha mixture (*see* p. 157).

Prognosis.—Where the general health can be rapidly improved and local treatment is instituted early, the

condition is soon got in hand. If, however, it is a complication of some serious blood disease or gross chronic infection it may terminate the illness more rapidly, usually giving rise to septic broncho-pneumonia.

Geographical Tongue

This is a common condition and seems to have little significance. The tongue shows areas where the surface is pink and normal, and these are interspaced by areas which are slightly raised and greyish in appearance. The shape of the normal areas may be round, or there may be a serpiginous edge to them, hence the name "geographical tongue." The cause of the condition is not clearly understood, but it is stated by some to be due to a low-grade chronic stomatitis. Certainly it is not as a rule accompanied by ill-health. It is stated by some authorities to be an accompaniment of the exudative diathesis.

Treatment.—There is no indication for treatment in this condition.

Caries in the Teeth

Dental caries is extremely widespread. In fact, it is the exception rather than the reverse to find a child whose teeth do not show signs of caries. The teeth of the hospital class of patient are still gravely neglected.

Evils of dental caries.—A carious first dentition will undoubtedly lead to caries in the permanent teeth. This is brought about by the poisons of decomposed food and bacteria passing directly from the temporary tooth to the permanent tooth below. In addition, when a permanent tooth appears in a mouth which contains carious teeth it is sure to become quickly infected.

A child with carious teeth is very much aware of its disability. It fails to chew on the side where the teeth are affected, as the slightest pain or discomfort is noted. The result is that food is swallowed in a poorly masticated condition and is consequently ill-digested and ill-absorbed, leading to *malnutrition*. The swallowing and absorbing of decayed food and bacterial debris which

collect in the teeth overnight give rise to a dirty tongue and chronic gastritis. In the author's experience he has rarely seen a perfectly healthy child with dental caries to any extent.

Nervousness can undoubtedly be traced in some children to bad teeth. This may be through lack of sleep and poor assimilation of food, but the most highly-strung, nervous children seen at hospital and those most difficult to examine have over and over again proved to have extensive dental caries.

Septic teeth infect the tonsils and give rise to *septic tonsils* with all the ills consequent on this condition. Septic teeth also cause *enlarged glands* in the neck, which in time tend to become tuberculous.

Etiology and pathology.—Teeth become carious in the first place because of deficient or badly-formed enamel. This is almost certainly the direct result of a rachitic diet, low in vitamin, badly balanced or short of calcium, and also of insufficient sunshine and fresh air. In addition the child may inherit a family tendency to poor bone-formation, and this may be an aggravating factor in the production of poor enamel.

A second factor is a failure to keep the teeth clean and in good condition, and as this lies entirely within the power of the child's parent it is the more culpable of the two.

Undoubtedly severe illnesses tend to leave permanent defects in the teeth. A great portion of the enamel of the teeth is laid down while the child is in utero, and therefore the food of the mother and her general health at that time may be of the utmost importance.

Another factor in the production of caries is an impaired saliva. The saliva normally contains bactericidal properties, and anything which impairs this allows the bacterial flora of the mouth to flourish, attacking the teeth.

Finally, if children are given soft and pappy meals with much sugar in them, and if sugar is given between meals, the proper jaw movements and the friction of hard food on the molars are prevented, and at the same

Plate 4.—Radiogram of cardiospasm, showing bismuth held up before entering the stomach by spasm of the lower end of the œsophagus.

time bacteria are allowed to flourish in the retained sugar lying in the interdental spaces.

Care of the teeth.—Every child should have the teeth cleaned by scrubbing with a soft, boiled tooth-brush twice daily from the age of 9 or 10 months. An alkaline mouth-wash, such as milk of magnesia, is sufficient, and as the child grows older the brush should be stiffer and more resisting. Hard foodstuffs, such as rusks, crusts and crisp toast, should form part of each meal, so that the gums are stimulated and the saliva is forced throughout the mouth. With the appearance of the smallest hole in either the first or second set of teeth, a dentist should be consulted, the hole cleared out and stopped. No amount of trouble is too great to preserve the first, as well as the second, set of teeth as long as possible. The removal of the first dentition for caries is most detrimental, as the tendency is for the jaw to fail to expand if the first teeth are removed early, and the result is overcrowding of the second dentition. No sweet food should be eaten before going to bed. The teeth must be cleaned the last thing, after all food is taken.

Cardiospasm (Œsophagectasia) [1, 2]

This is a very rare condition occurring in infants and young children and only with difficulty distinguished from true œsophageal stricture due to some congenital malformation or acquired by trauma.

Symptoms.—The child is as a rule under-nourished and under-sized, but occasionally is normal in appearance. Once or more times in the day vomiting occurs, especially at any attempt to take solids. Fluids are better retained. A bismuth meal may pass rapidly into the stomach, but as a rule the X-rays give a picture similar to that shown in Plate 4. The site of true cardiospasm is most commonly at the extreme lower

[1] Donald Paterson, "A Case of Œsophagectasia in a Child," *Brit. Journ. Child. Dis.*, Jan.-March, 1921, Vol. 18, pp. 27-29.

[2] Wilfred Sheldon and A. G. Ogilvie, "Obstruction of the Œsophagus in Childhood," *Arch. Dis. Child.*, 1929, Vol. 4, p. 347.

end of the œsophagus, there being a dilatation or pouching of the œsophagus.

Etiology.—Whether there is a primary ulcer with resulting spasm of the muscle due to a stimulus of the nervous control, as Cameron states,[1] it is impossible to say. Certainly the condition is intermittent, and there are bad periods when the spasm is present and vomiting is frequent, then periods, sometimes of many months, when the child appears normal. The condition may persist into adult life.

Treatment.—Just as in adults, the treatment in children is extremely satisfactory. Dilatation of the narrowed portion of the œsophagus by means of a rubber tube filled with mercury is very successful. In a recent case aged eight under the author's care at the Westminster Hospital, the child gained several pounds and his vomiting completely ceased after passing a mercury tube before meals. This he did himself. He was sent home with the tube, which he continued to use.

Œsophageal stricture or stenosis.—In this condition the stenosis may be complete, the œsophagus terminating in a blind end at the level of the bifurcation of the bronchi. In a case of this kind vomiting occurs from birth and there is rapid wasting resulting in death. Where there is merely a narrowing the child may live on for many years, being under-sized and able to take thin fluids but no solids. Such cases are continually examined for cardiospasm, dilatation being attempted with bougies and mercury tubes, with or without anæsthesia. The author's experience is that such cases ultimately do very badly, although with occasional dilatation they are improved.

Where the stricture is congenital the site is almost invariably as stated above. Where it is acquired from trauma with some corrosive the ultimate prognosis may be much better, although there is a tendency in future years for the stricture to become progressively worse.

[1] J. A. M. Cameron, "Œsophagectasia in a Child," *Arch. Dis. Child.*, Vol. 3, p. 358.

Congenital Pyloric Stenosis, [1, 2, 3, 4]

As the name indicates, this condition exists at or before birth. The sphincter or muscle guarding the exit from the stomach is unusually thickened, with the result that food is prevented from leaving it. Symptoms of obstruction then occur.

Sex-incidence.—Pyloric stenosis is much more common in males than in females, with a ratio of 6 or 7 males to each female.

Age-incidence.—The initial symptoms occur most commonly at the end of the second week after birth. Occasionally, however, the vomiting commences at birth or as late as the tenth week. The condition is most common in the first children of young mothers. The mother is generally of a highly nervous type and most intelligent.

Pathology (based on more than 50 post-mortem examinations).—The tumour is firm, round or oval in shape, and the muscle-fibres on incision are found to be hard and in some cases feel almost cartilaginous. Usually there is asscciated gastritis with this condition. Microscopically the circular muscle-fibres of the pylorus are seen to be greatly hypertrophied and thickened. There is no doubt that the whole of the stoppage or stenosis cannot be accounted for by thickening of the pyloric ring, and it is the author's opinion that in all cases there is superadded spasm. The reason for this view is that these children undoubtedly do pass stools, and when a certain degree of debility occurs death is usually due to diarrhœa. The generally accepted theory of the causation of the condition is that the hypertrophy is due to an inco-ordination of the gastric and intestinal peristalsis. Very few cases of pyloric stenosis have been recorded at birth, although many cases show clinically symptoms of pyloric obstruction at this age.

[1] Gray and Pirrie, *Lancet*, 1919, ii. 515.

[2] Poynton, Higgins and Brydson, *Lancet*, 1924, i. 215.

[3] Still, *Brit. Med. Journ.*, 1923, i. 579.

[4] Paterson, "Congenital Pyloric Stenosis," *Nursing Mirror*, Feb. 20 and 27, 1926.

Symptoms and diagnosis.—As a rule the infant is breast-fed and seems to thrive up to the age of two weeks, when vomiting commences. This is of the projectile type, and the vomit is large and obviously consists of more than one feed. It contains curdled milk and mucus, and is occasionally tinged with blood. A day may pass with only one vomit, but the next day several vomits immediately following on the feeds are the general rule. The infant wastes but remains surprisingly bright. He has an eager, hungry look and gnaws at his hands. Coincident with the vomiting marked constipation occurs. As a rule the vomiting is put down to the food being unsuitable, and a new food or method of feeding is tried, with, for a day or two, what appears to be success; then the vomiting recommences. The diagnosis rests on:

1. The history of persistent large vomits.
2. Obstinate constipation.
3. Seeing vigorous gastric waves passing from left to right across the abdomen (Fig. 22).
4. The palpation of a pyloric tumour.

Method of diagnosis.—When presented with a case of persistent vomiting with obstinate constipation pyloric obstruction should be suspected; the child should be given a feed and gastric waves watched for. The mere "standing out" of the stomach is insufficient, as this occurs in so called pylorospasm. Waves the size of a small tangerine orange may be seen passing from left to right across the abdomen. Very often two waves may be seen at the same time. The more wasted the child the better the peristalsis. The waves must be vigorous, and in every case of true pyloric stenosis this will be the case if the infant is observed for any length of time. During the feed, if the abdomen is palpated, a pyloric tumour can almost always be felt—sometimes superficial, sometimes deep in the abdomen, immediately to the right of the right rectus, high up and just beneath the liver margin.

The only two conditions which in any way simulate

pyloric stenosis are : (1) congenital stoppage or stenosis of the duodenum, and (2) pyloric spasm.

Fig. 22.—Congenital pyloric stenosis, showing peristaltic waves passing across the child's stomach.

In congenital stoppage of the duodenum vomiting always commences at birth. Constipation is obstinate or complete. The vomit contains bile and is green in

colour. Gastric waves occur but no tumour can be felt. As many girls as boys are affected. Resort to X-rays settles the diagnosis definitely.

With cases of pyloric spasm insufficient feeding or a tendency to swallow air is most common. The vomiting is intermittent and there may be remissions of several days, constipation being similar to but much less persistent than that in stoppage of the duodenum, the child having normal stools or even small bouts of diarrhœa. Gastric waves are never vigorous.

Treatment.—The diagnosis having been definitely made, one must choose between medical and surgical treatment. To make the diagnosis certain, however, may require several days, and during this uncertain period resort to gastric lavage relieves the child of the retained food and tends to prevent gastritis. Subcutaneous or rectal salines should be given if the child is markedly wasted.

Medical treatment.—For a number of years this was carried out more or less successfully, but with the introduction of operation it has largely fallen into disuse in this country. Nevertheless, on the Continent, especially in Scandinavia, medical treatment is still persevered with, and in one clinic in Copenhagen 98 per cent. recoveries are recorded. The treatment consists of gastric lavage (*see* p. 168) two or three times daily, with careful feeding. The feed chosen is usually one such as peptonized milk or breast-milk, where the curd is extremely small and can pass through a small opening of the pylorus. Small doses of belladonna or atropine help to relax the spasm. Fresh air and sunshine are useful adjuncts, although the utmost care must be taken to prevent chilling the infant. In this country results from medical treatment, even under the best circumstances, are not good. When a cure takes place, the weight of the child is maintained stationary until the fourteenth or fifteenth week has been reached, at which time a sudden improvement occurs and recovery takes place in a dramatic fashion. To secure this, however, the utmost care in nursing is essential, and both a day-

and a night-nurse are required who are able to perform gastric lavage. During these trying weeks a slight chill, giving rise to pneumonia, or a temporary relaxation of the pylorus with a short bout of diarrhœa, may end the struggle unfavourably. *The author, personally, prefers surgical treatment.*

Surgical treatment.—The essential for this is that the cases should be secured early. When the child is two, three or four weeks old, and especially when breast-fed, the mortality is practically nil. At the eighth or tenth week, and when he is artificially-fed, the outlook is by no means so bright. Two days' preparation is, as a rule, necessary. The stomach is washed out twice daily. The first day 3 or 4 ounces of saline should be given into the axillæ or groins subcutaneously, and the second day 2 to 3 ounces of the parent's blood, intravenously when possible. If the transfusion is not possible a second saline should be given. No atropine injection is given prior to operation, but the stomach is washed clean with normal saline an hour before it takes place. The arms and legs of the infant should be enveloped in cotton-wool, lightly bandaged on to prevent cooling, and when the child is on the operating table the hands may be fastened to the pillow. Gas-and-oxygen is the best general anæsthetic. Recently local anæthesia has been used very successfully in these cases.

Rammstedt's operation consists of splitting the circular fibres of the pylorus so that they spring apart like the tyre on a wheel, allowing the food to pass through. It is a short operation requiring less than 10 minutes. The infant when returned to its cot must be kept very warm. For the next six hours its rectal temperature should be taken hourly to prevent a hyperpyrexia, in which case bottles and covering should be removed and an ice-bag placed on the head.

Post-operative feeding.—This consists in giving one-drachm feeds of either breast-milk, half-cream dried milk, an unsweetened condensed milk mixture (*see* p. 57), or a peptonized milk and water mixture at

hourly intervals, commencing 6 hours after the operation. Twelve hours after the operation the amount is increased to 1½ drachms hourly. Eighteen hours after the operation 2 drachms are given every 1½ hours, and the amount is gradually increased so that 24 hours after the operation the child is getting ½ oz. every two hours. If there is an adequate supply of breast-milk, the infant should be allowed to nurse at the breast three-hourly from this time onwards. If not breast-fed, 36 hours after the operation the amount is increased so that the feed is 2 oz. every three hours. By 48 hours after the operation the feed given is almost the full amount for the infant's weight. If breast-milk is not available, an unsweetened condensed milk mixture is preferable as first choice. It is made in this way: unsweetened condensed milk one part, water 4 parts, Demerara sugar one heaped teaspoonful to each 4 oz. of the mixture. Peptonized milk mixture should be made of equal parts of milk and water, and peptonized for 30 minutes. A heaped teaspoonful of Demerara sugar should be added to each 3 oz. of the mixture.

After the operation one or two subcutaneous salines may be found necessary. If vomiting recurs the stomach should be washed out twice daily. At any sign of diarrhœa the sugar should be reduced and the feed lessened for the time being. Rapid gain in weight should be expected between the fourth and seventh day.

It is interesting to note that these children ultimately become strong and healthy.

Gastric and colonic lavage.—In cases of dyspepsia or vomiting it is often necessary to *wash out the infant's stomach*. This is not difficult, a soft, small, red rubber œsophageal tube, with a glass funnel attached to the open end, being used. It is also possible to use the barrel of a 20-c.c. syringe instead of the glass funnel. The outside of the tube should be moistened at the beginning of the process, so that the tube can be passed into the baby's stomach without the use of any lubricant. If much mucus is present a solution of sodium bicarbonate (one drachm to the pint) may be

helpful instead of water. Saline should be used instead, however, when there is severe vomiting.[1] The liquid should be warmed, and the infant's stomach must not be over-distended by using more than a few ounces at a time. By alternately raising the funnel above and then depressing it below the level of the infant, fluid may be run into or out of the stomach, and this process should be continued until the fluid is returned clear and unaccompanied by food debris or mucus.

A similar apparatus may be used for *washing out the rectum* and lower few inches of the colon, and warm saline is preferable to ordinary water. A pillow is covered with a mackintosh sheet and the buttocks are raised over this. The well-greased tube is then inserted gently to about 1 or 2 inches, and to prevent over-distension the level of the funnel should not be more than 18 inches above the infant. No fluid is likely to be retained when the operation is finished.

Duodenal Stenosis and Atresia

Atresia may be due to some developmental error in the gut itself, associated with partial or complete absence of its lumen. Stenosis is commonly due to compression or constriction of the duodenum by abnormal peritoneal bands or adhesions; in this case the gut possesses a normal structure, but its lumen is partially or completely obliterated (Fig. 23).

Symptoms.—Where atresia occurs vomiting is marked, especially of bile-stained fluid. Constipation is complete, and material obtained by washing out the lower bowel shows meconium only. Where the atresia is partial only, very small quantities of food may find their way through, the bowel being narrowed for a considerable length, sometimes throughout the whole of the small intestine.

The commonest form of duodenal stenosis is due to bands or adhesions. In such a case the clinical picture is extremely like that of congenital pyloric

[1] Maizels, McArthur and Payne, "Alkalosis in Congenital Hypertrophic Pyloric Stenosis," *Lancet*, Feb. 8, 1930, p. 286.

stenosis, there is marked vomiting with constipation, the infant fails to gain weight and is restless and crying. The vomit contains bile as a rule, thus differing from that of pyloric stenosis. On examination waves can be seen passing from left to right across the epigastrium, but they pass far out into the right flank. The pylorus cannot be felt in such cases. It may be necessary to

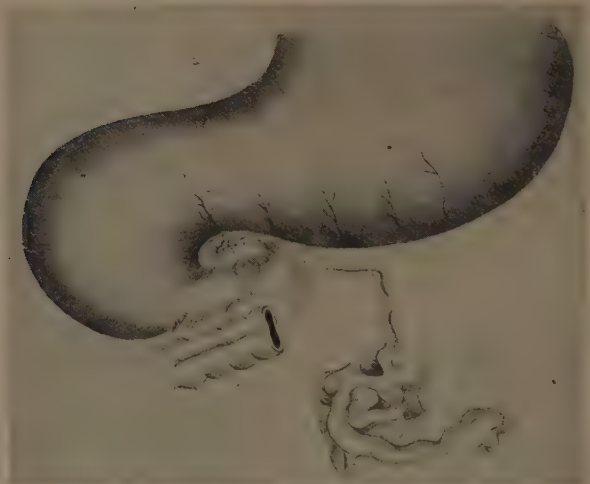

Fig. 23.—Congenital duodenal stenosis, showing distension of stomach and first part of duodenum.[1] (*Archives of Diseases in Childhood.*)

give some bismuth to complete the diagnosis, when the duodenum is shown by X-rays to be greatly dilated throughout, as in Fig. 23.

Prognosis and treatment.—The outlook in true congenital atresia is extremely bad, for when the bowel ends blindly, or when there is a narrowing over any considerable length of bowel, the difficulties before the surgeon appear almost insurmountable. The cases

[1] T. T. Higgins and Donald Paterson, "Congenital Duodenal Stenosis due to Peritoneal Bands," *Arch. Dis. Child.*, Oct. 1926, Vol. 1, No. 5. (These authors give a bibliography of the whole subject.)

which react best to surgical treatment are those in which the stenosis is due to bands or adhesions, and several recoveries have been reported. Gastro-enterostomy may be necessary where the bands cannot be freed. In every case a laparotomy should be performed and the child given the advantages of surgical treatment.

Duodenal Ulcer

This condition is probably much commoner in infants than is generally realized, but must still be regarded as very rare. It is occasionally seen in the first few months of life, associated with wasting and accompanied by melæna. The child as a rule is blanched and pale and there may be an extreme degree of anæmia. Pain is seldom complained of and the temperature is usually normal. The pulse-rate, however, is rapid. The author has seen two cases, of which Fig. 24 is a good illustration.

Fig. 24.—Duodenal ulcer in a child aged 3 months.[1] (*Lancet.*)

1, Stomach; 2, pylorus; 3, duodenal ulcer; 4, ampulla of Vater.

[1] Donald Paterson, "Duodenal Ulcer in Infancy," *Lancet*, 1922, i. 63.

Treatment.—This should be by transfusion and small frequent feeds of peptonized milk. Rectal feeding should be instituted as far as possible. Alkalis by the mouth are indicated. One case in which the diagnosis was made was successfully operated on, a gastro-enterostomy being performed.

Appendicitis[1]

This is the most common, and may be the most serious, of all the inflammatory abdominal conditions. Early diagnosis is of the utmost importance.

Etiology and pathology.—Appendicitis may occur at any age, but is uncommon before the age of three. There is no sex predominance. By some it is claimed to be due to an infection gaining entrance to the blood-stream, possibly through the mucous membrane of the bowel or through the tonsils, and passing directly to the appendix. Others contend that it is a local abscess, starting in the lymphoid tissue of the appendix, due to kinking or blocking of the appendicular lumen. Certainly threadworms filling the appendix would give rise to a typical attack. An association between appendicitis and rheumatism was pointed out many years ago by Poynton and Paine.[2] There is ground for believing that there may be a specific infection in many cases, as in schools and institutions epidemics of this complaint are not uncommon. Once the organ has become inflamed it may be distended and congested, this inflammation extending through the walls to the peritoneal coat and setting up local or general peritonitis. With a very virulent or severe infection, local gangrene may develop with perforation, the fæcal contents of the appendix finding their way into the abdomen directly. Where the omentum becomes

[1] L. E. Barrington-Ward, "The Abdominal Surgery of Children" (Oxford Medical Publications), p. 121.

[2] Poynton and Paine, *Lancet*, 1911, ii. 1889.

adherent in the region of the appendix, the whole may be sloughed off and an abscess may form.

Symptoms and diagnosis.—The chief symptoms are abdominal pain accompanied by vomiting, with tenderness and rigidity of the lower right rectus (McBurney's point). The clinical picture varies with the site of the appendix and the stage of the disease. Where the appendix is retrocæcal the clinical picture is often atypical, there being pain and tenderness, but not to the same extent as in the average case, and vomiting is unlikely to be a feature. Often the symptoms are so slight that they are taken for an ordinary bilious attack. Fever is not constant, but is usually present, especially in the smaller children. In every case of doubt a rectal examination should be made. In doubtful cases the very greatest importance should be attached to the time of onset of the pain. If this precedes the vomiting, it is of the greatest significance; following on vomiting, especially if much retching has occurred, it is not of so much clinical importance. Tenderness and rigidity or spasm of the abdominal muscles, coupled with abdominal pain, make the diagnosis complete.

The appearance of the patient is of the greatest significance, the eyes being sunken and the tongue furred and dry. The pulse is raised out of proportion to the degree of fever, if any, and is bounding. As a rule constipation is obstinate, but occasionally there may be a tendency to small frequent motions.

Differential diagnosis.—Usually the diagnosis is easily made, but occasionally it may be mistaken for a *bilious attack*. In such cases the absence of abdominal pain with tenderness and rigidity should be of some help. In the retrocæcal type, however, there is a close similarity between the two and a rectal examination alone will decide the diagnosis. *Diaphragmatic pleurisy* and *right-sided pneumonia* may present serious difficulties in the early stages. The general appearance of the child should be of the greatest help, as the face is flushed and the respirations are greatly increased.

A rectal examination does not show the tenderness commonly found.

Inguinal and mesenteric adenitis, when the deep group of glands in the inguinal region on the right side become inflamed, may be difficult to distinguish from appendicitis. The general systemic upset to the child is less marked as a rule and colicky pains tend to pass off rapidly. Fortunately this condition is rare. *Pyelitis* in the right kidney may simulate appendicitis closely, but an examination of the urine should settle this point at once. *General peritonitis due to the pneumococcus* may present the same difficulty. In this condition tenderness is present throughout the whole abdomen, toxæmia is extreme, and pain has not been limited at any time to the right iliac fossa. It is only by local examination and consideration of the child as a whole that a differential diagnosis can be made. Inserting a needle into the peritoneal cavity has been suggested by some observers to decide the diagnosis. *Acute follicular tonsillitis* is sometimes accompanied by colicky pains about the umbilicus (*umbilical colic*). The mode of production of these pains has not been satisfactorily explained, but they are universally recognized. With the subsidence of the tonsillitis the abdominal pain disappears.

Prognosis.—The sooner the diagnosis is made and treatment instituted the better the prognosis. Once general peritonitis has occurred the mortality rises very rapidly, especially in children under the age of six. With early diagnosis and efficient surgical treatment the mortality should be nil.

Treatment.—The treatment is purely surgical.

CHRONIC OR RECURRENT APPENDICITIS

The question whether true chronic appendicitis occurs in childhood is debatable. Probably the term *recurrent appendicitis* is much nearer the truth, but even recurrent appendicitis must be considered an extremely rare condition.

Symptoms and clinical picture.—After a mild or sub-acute attack of appendicitis, with fever, pain, vomiting and tenderness, the symptoms may subside and the whole illness may be regarded as merely a bilious attack. Some weeks or even months later there may be a recurrence, this time possibly with sharp, stabbing pains or nausea, dirty tongue, lassitude and a low, "grumbling" temperature. The temperature may never have properly settled between the first attack and the second, although on examination between the exacerbations there is little or nothing to be found. Such recurrences may go on for some months or even years before the child is seen in one of the attacks and a diagnosis is made.

Differential diagnosis.—Some of the conditions which may simulate recurrent appendicitis are inflamed abdominal glands, tuberculous or otherwise, intermittent hydronephrosis, calculus, adherent Meckel's diverticulum, a partial volvulus, chronic intussusception, or follicular tonsillitis with so-called umbilical colic and acidosis attacks, or dragging on the mesentery in a nervous child. It is only by keeping such conditions as these in mind and excluding each when possible that a correct diagnosis can be arrived at.

X-rays.—In a series of suspected cases submitted for X-ray examination, no very real help can be said to have been obtained. The bismuth certainly passed readily into the majority of normal appendices, but where it did not, no definite evidence of disease was necessarily found at operation.

There is a body of opinion which regards so-called acidosis or cyclical vomiting attacks as merely recurrent appendicular attacks, and the removal of the appendix is done by some as a routine. Unfortunately, although occasionally the appendix is the source of the trouble, its removal does not constitute a cure in such cases, and the author has known of many patients complaining of the same symptoms after operation.

Very often the question of appendicitis is raised on account of abdominal pain and this gives rise to

constant fear in the mother's mind that it may recur unnoticed. In these circumstances although the diagnosis may not always be absolutely clear, the removal of the appendix would appear to be justified in order to set the mother's mind at rest.

In cases of true acute appendicitis, where there is acute inflammation and later abscess formation, there is seldom if ever a history of previous appendicitis attacks. There would seem to be little evidence, therefore, for believing that true chronic appendicitis does exist in childhood. Recurrent appendicitis or, to use a much better term—subacute appendicitis, which it should be possible to diagnose if seen in the acute stages, is the equivalent in childhood of adult chronic appendicitis.

Chronic constipation in childhood, giving rise to vague abdominal pain, and recurrent severe colic when purges are used to get the bowels to move, is often mistaken for recurrent appendicular trouble. Such a child, put on a proper régime with suitable treatment rapidly loses his symptoms (*see* pp. 135-140).

Pneumococcal Peritonitis

There may be a primary infection of the peritoneum with the pneumococcus or an infection secondary to some other pneumococcal process in the body.

A. primary pneumococcal peritonitis

This is a disease which as a rule affects females only, especially between the years of three and seven. It is much commoner among the poor than the well-to-do. It has been clearly shown by McCartney and Fraser[1] and others that the majority of the patients have previously suffered from vulvo-vaginitis and that virulent pneumococci may be obtained from the vagina in such cases. The infection starts in the pelvis and is almost

[1] J. E. McCartney and John Fraser, "Pneumococcal Peritonitis," *Brit. Journ. of Surgery*, Vol. 9, No. 36, 1922, pp. 479-489.

certainly the result of an ascending infection from the vagina, through the genital tract.

Clinical picture.—Both as a preliminary and accompanying the onset of the infection, there is much diarrhœa. Abdominal pain varies and may be most misleading. It is on the whole correct that the pain in such cases is much less than would be expected; it may be slight or entirely absent. The onset is acute, with high fever and delirium suggestive of pneumonia. It is probable that at this stage there is a septicæmia. Later there is abdominal discomfort or pain, with a gradually increasing tumidity of the abdomen. On palpation there is tenderness of the abdomen. The temperature continues high for some days and tends to settle gradually. The abdomen, which has been full and tumid, gradually becomes more doughy, and loculated areas of pus may be made out. The intestines are matted together and may show ladder patterns.

Differential diagnosis.—The acute onset may suggest appendicitis, and it is with this in mind that operation is generally undertaken. Again, if the condition has gone on for a few days the picture may somewhat resemble typhoid fever, with delirium, a tumid, slightly tender abdomen and diarrhœa. The sudden onset, the delirium and the presence of pus in the peritoneal cavity, and the fact that the patients as a rule are girls, should point to the correct diagnosis.

Treatment.—It is most debatable whether operation should be undertaken or not. McCartney and Fraser advocate immediate operation. Bellingham Smith[1] and others believe that operation should be left until the septicæmia stage is over and a residual abscess has formed in the peritoneal cavity. This is probably the safer course. Blood transfusions are in the author's opinion the most useful form of treatment, with laparotomy in the loculated stage.

Prognosis.—Those cases in which a residual abscess forms recover completely when this is drained. Cases

[1] *Proc. of the Roy. Soc. Med.*, Discussion on Pneumococcal Peritonitis, Vol. 18, No. 7, May 1925

with a high fever and what appears to be septicæmia stand operation badly and very few seem to recover. The mortality is about 50 per cent.

B. SECONDARY PNEUMOCOCCAL PERITONITIS

This may follow on a pneumonia and is included among the possible complications of pneumonia. It may be accompanied by pneumoccocal infections of other serous cavities, such as the middle ear and the pericardium. When there is a general pneumococcal infection the prognosis is extremely grave. If, however, following on a primary pneumonia, peritonitis appears somewhat later and the pneumonia clears up, the peritonitis may be dealt with as a residual abscess and drained, and recovery may be expected.

Colitis

Acute entero-colitis or acute infection of the intestinal tract with some pathogenic organism has already been mentioned in the description of infective diarrhœa (p. 117). The characteristic of this condition is the presence not only of mucus but also of blood in the stools, and at post-mortem there is often a definite ulceration of the mucous membrane of the bowel.

MUCOUS COLITIS

Mucous colitis may be present in infants who suffer from diarrhœa during dentition. It may also alternate with eczema in fat, watery, eczematous babies, and it is characteristic of the so-called *exudative diathesis* child to have bouts of loose mucus-containing motions at intervals.

The condition is seen most characteristically in older children. In the author's opinion there is a very close connexion between mucous colitis and so-called acidosis attacks.

Etiology.—The primary source of the trouble is usually some acute infection, more often than not a

tonsillitis, and the child, with its unstable metabolism, rapidly forms acetone and loose stools follow. Catarrhal mucous colitis may follow on overfeeding with fats, so that an excess of uncombined fatty acids is present in the motions, causing the stools to be strongly acid, and thus burning or excoriating the bowel mucous membrane. An excess of carbohydrate or sweet or starchy foods similarly produces a fermentative acid diarrhœa, and consequently a catarrh of the bowel-lining. In the same way doses of castor oil for constipation provoke in the child a similar catarrh, and should be avoided as far as possible.

Clinical picture.—At recurrent intervals the child has bouts of fever with offensive, acid, mucus-containing motions, but seldom if ever is blood present. These bouts become steadily less frequent as the child gets older. Examination of the motion shows long strings of jelly-like or membrane-like mucus—in fact it looks as if the lining of the bowel were being shed. Actually the strings consist of dried mucus. The whole attack may last two or three days, or even a week, and then gradually subside. Accompanying it there may be a slight sore throat or a flare up of some septic focus. There are cases, however, in which the trouble would appear to be primarily dietetic. Any slight indiscretion, especially in fat (milk, cream, butter and eggs), brings with it this train of symptoms. Probably the commonest cause, apart from infections, which the author has encountered in actual practice is giving the child an excess of rich milk. Very often between attacks a child may receive as much as a quart of milk per day, with the result that he has recurrent bouts of loose, acid motions, light in colour and containing much mucus. As in the so-called "cyclical-vomiting" child, so in these cases, maternal anxiety and faulty régime are of great etiological importance.

Treatment.—The diet should be a well-regulated one with a low fat-content, such as that given on p. 134. It may be necessary, however, to sieve all vegetables and avoid raw fruit (except orange juice) until the

bowel has recovered its normal condition. Care should be taken not to exceed two eggs in the week nor half a pint of skimmed milk per day.

During the actual attack a small dose of castor oil should be given, followed by a diet of bland fluids, such as drinks of orange-water, sugar-water or barley-water. Arrowroot made with water, or cornflour, or Robinson's Groats, or Benger's Food made with equal parts of skimmed milk and water, should next be attempted. No fresh cow's milk should be given if it can be avoided. A little Horlick's or Hooker's Malted Milk or Half-Cream Cow and Gate may be substituted with advantage. A gradual resumption of ordinary food, returning to milk and eggs last, should be made. Starchy foods suit such cases best as a rule. The régime suggested should be that for a child with cyclical vomiting or fat indigestion (*see* pp. 128, 132).

Intussusception

Etiology.—Intussusception is slightly more frequent in boys than in girls, and is commonest between the ages of 6 months and 18 months, although it may occur at any age. The patients are usually vigorous, strong children. At a children's hospital it was noticed that intussusception generally appeared at week-ends and during the holiday season, and food indiscretions have long been considered the primary cause, acting, it is thought, by causing a gastro-enteritis which inflames the lymphoid tissue, especially about the cæcum. The nodule of such lymphoid tissue serves as a starting-point from which the intussusception moves forward. A polypus or other growth will also act in the same way.

Various forms.—The most common is the ileo-cæcal form, in which the ileum is invaginated into the cæcum, and passed up the ascending colon and onward. The next most common is the ileo-colic, then the purely enteric, or intussusception of the small intestine only. Finally there is the purely colic type.

Clinical picture.—A fat, well-nourished child, usually breast-fed, between the ages of 6 and 9 months, suddenly screams and draws up its legs, looking grey and ill. The attack may pass off, to be followed quickly by another and yet another at varying intervals of a few minutes. Very often this is followed by a stool containing blood and some mucus, which may be repeated. No further fæces are passed. Between attacks of pain the child may appear quite bright, although not himself. Careful palpation of the abdomen reveals a sausage-shaped mass, usually lying transversely across the abdomen. The mass may be soft and difficult to feel, but with continued palpation it hardens under the hand, so that it is unmistakable. On rectal examination the end of the mass may be felt, if it has travelled round to the sigmoid, but usually this is not the case. On withdrawal, however, the finger is covered with blood and mucus.

Prognosis.—Taken early the outlook is excellent, but the mortality rises steeply after 12 hours. After 24 hours the outlook is bad.

Differential diagnosis.—The commonest mistake is to confuse an acute entero-colitis with intussusception. In the former, blood and mucus are present in the motions, the child is collapsed and there may be vomiting. Attacks of pain are present, but not so severe nor so frequent. The temperature is high. An examination reveals no mass and the motions are frequent, although small. Other conditions to be excluded are Henoch's purpura and abdominal tuberculosis.

Treatment.—The treatment is surgical and should be proceeded with at once.

Chronic intussusception.—This usually occurs in older children, but may be found in infants. A child may have attacks of abdominal pain at hourly intervals for some weeks, and on examination a mass is to be found. Meanwhile motions are passed and blood is unlikely to be present. With operation and reduction such cases tend to recur.

Volvulus

This is an extremely rare condition in childhood. It may occur at any age.

Etiology.—It is due either to some congenital defect of the mesentery—malrotation or hernia of the bowel through an incomplete mesentery, bands, etc., or to an acquired defect such as an adherent Meckel's diverticulum.

Clinical picture.—The classical symptoms of obstruction are usually present, vomiting, pain, collapse. Occasionally there are recurrent cases which undo themselves and pass for bilious or acidosis attacks. In such cases, however, the abdomen is full and distended rather than scaphoid, as is the case in a true bilious attack.

The **treatment** is surgical.

Catarrhal Jaundice, or Acute Hepatitis [1]

Etiology and pathology.—This may occur at any age, but is commonest between 5 and 8 years. Since the child invariably recovers, no good pathological evidence about this disease is available, but it should be considered an acute infection of the liver-cells, with catarrh of the bile canaliculi. Very often the infection is an ordinary influenzal cold, and more than one child in the family may be affected. Invariably, in the author's opinion, some dental or nasopharyngeal sepsis is found and is likely to have played an important part in the causation of the attack.

Clinical picture.—Catarrhal jaundice commences with vomiting and occasionally diarrhœa, with a greater or less degree of fever. It is variously named at this stage "gastric influenza" or "chill on the liver." The child is drowsy. In from two or three days to a week the jaundice gradually appears, the liver being enlarged sometimes to the umbilicus and the spleen being almost invariably palpable at some time during

[1] Robert Hutchison and Donald Paterson, "Infective Hepatitis with Cirrhosis," *Brit. Journ. Dis. Child.*, Vol. 21, pp. 275-279.

the course of the disease. The sclerotics show jaundice first and bile is present in the urine. Gradually the stools become lighter and lighter until they are white or clay-coloured, the skin becoming a dark yellow-brown. Once the jaundice is well developed, as a rule the vomiting and drowsiness with temperature clear up, and the child feels quite well in himself. The tongue remains dirty and the stools colourless for some days. It may take two or even three weeks for them to return to normal, and this should coincide with the disappearance of the jaundice from the skin and sclerotics.

Complications.—Where there has been a recurrence of the catarrhal jaundice on more than one occasion or when, in what appears to be an ordinary attack, the temperature has not settled and the symptoms continue to get worse, one may suspect that the liver infection is not a simple catarrhal one, but is of a much more severe type. All such cases should be looked on as very grave, as they may be the forerunners of *acute or sub-acute yellow atrophy.*

Treatment.—Every patient with catarrhal jaundice should be put to bed and kept there until the condition has completely cleared up. In this way serious complications may be avoided. If the bowels are constipated small doses of grey powder or calomel should be given, as they also stimulate the liver.

Diet.—It is most important to put the child on a low-fat, low-protein, high-sugar diet. Proteins are de-aminated in the liver, throwing work on it, and fats are also dealt with in that organ. The carbohydrate alone can be absorbed and utilized without the aid of the liver, and it is on the starches and sugars that the child must depend for nourishment, during both the acute illness and convalescence. If fats and proteins are prematurely reintroduced into the diet a return of the jaundice is most likely to occur. Cereals for breakfast with fresh or stewed fruit, vegetables and gravy followed by stewed fruit for dinner, and some cereal or crisp toast with weak tea at tea-time will

be found suitable for such cases. As the colour returns to the stool a less rigid diet is possible.

After-treatment.—The removal of septic teeth and tonsils is a great help in the prevention of further liver infection.

Acute or Subacute Yellow Atrophy

This is an extremely rare disease which starts like an attack of ordinary catarrhal jaundice, the symptoms, however, being much more severe, with headache, delirium, vomiting, diarrhœa and high fever. There may be two or three attacks of what appears to be catarrhal jaundice preceding the onset of the severe liver infection.

Etiology.—The disease is commoner in older children and young adults. The cause is unknown, but certainly septic tonsils and bad teeth are present in a number of cases, and others follow on anæsthetics or poisoning with phosphorus, etc.

Pathology.—The liver-cells show acute necrosis, with an attempt, if the case lasts any length of time, at regeneration, a budding out of the liver-cells being most noticeable in the region of the bile canaliculi.

Clinical picture.—The child, who has been drowsy and vomiting, becomes gradually delirious and jaundiced, the urine being deeply bile-stained and the motions pale, with diarrhœa. What was at first thought to be catarrhal jaundice is now seen to be a grave illness. Delirium is a feature. The liver, which at first was quite palpable, shrinks rapidly beneath the costal margin and the spleen cannot be felt. There is a tendency for hæmorrhages to occur in the mucous membranes and purpuric patches to appear on the skin. Air-hunger and coma rapidly supervene, death taking place in the severe cases.

Prognosis.—Patients who survive are left with cirrhosis of the liver and are often said to have suffered from a severe catarrhal jaundice attack, but in the author's opinion catarrhal jaundice merges by degrees

into so-called acute yellow atrophy and there would seem to be a difficulty in drawing any distinction between the two, except in the prognosis.

Cirrhosis of the Liver [1]

Apart from congenital obliteration of the bile-ducts, which is mentioned on p. 51, various other cirrhoses

Fig. 25.—Hepatic cirrhosis in a child aged 6½.

occur throughout childhood. On the whole, however, liver cirrhosis may be said to be extremely rare and to be uncommon before puberty. Banti's disease, which would seem to occur between 10 and 11 years of age, is mentioned on p. 233. Infective cirrhosis, following on a severe so-called catarrhal jaundice attack, has already been mentioned (*see* p. 182).

[1] F. J. Poynton and W. G. Wyllie, "Hepatic Cirrhosis in Children," *Arch. Dis. Child.*, Vol. 1, No. 1, p. 1.

Fig. 26.—Case of cirrhosis of the liver with ascites (p. 185).

Prolapse of the Rectum

Incidence.—Prolapse of the rectum is uncommon and is not seen more often in one sex than in the other. It may occur at any age, but is most frequent between the ages of one and three. The author has seen a case aged six weeks.

Etiology and pathology.—Contrary to expectation, diarrhœa accompanies the prolapse more often than constipation, and wasting is therefore an almost invariable accompaniment also. This is frequent in cases of cœliac disease or chronic intestinal indigestion. With constant frequent stools there is much loss of the intrapelvic fat, especially in the ischio-rectal region. The rectum is not supported properly, the sphincter becomes lax and there is a protrusion at the anus, at first very slight, and finally a large extrusion as big as a tangerine orange may be seen. Bleeding from

this surface and ulceration are common, and there may be great difficulty in replacing the mass.

Treatment.—The mass may be replaced by covering it with a piece of clean lint or muslin, placing the whole hand over it and gently pressing. The child's attention should be distracted if possible by talking to him or interesting him in food or some other object. If necessary an anæsthetic may be given.

Once the mass is replaced, the buttocks should be strapped tightly together, the child lying fully extended. The ankles and knees should also be bandaged, and it is most useful if a long Liston splint can be bandaged to the side of the child to prevent him sitting up. The bowels should be kept well open and the motion passed in this recumbent position, thus preventing great straining and a recurrence of the prolapse. This treatment should be continued for from one to two weeks, allowing the sphincter ani to recover its tone. Treatment should be given at once for the underlying wasting. In a case of cœliac disease, dieting on the proper lines should be instituted (*see* p. 115), and if the patient is merely a badly-fed, poorly-nourished child, a proper diet and tonics should be administered.

Alcohol injection.[1, 2]—Probably one of the most efficient methods of dealing with rectal prolapse is by alcohol injections. This is an extremely simple matter and cannot be termed a surgical operation. It is to be done by the physician. The bowel is carefully washed out as a preliminary and the child is given a general anæsthetic. The skin of the buttocks and anus is cleansed with ether soap and spirits. A fine needle some 3 in. long is obtained with a small Record syringe containing 1 c.c. of absolute alcohol. The finger is inserted into the anus and the point of the needle is inserted just to the outside of the point where the anal mucous membrane and the skin meet, that

[1] Alan Brown and T. G. H. Drake, "Treatment of Prolapse of Rectum by Alcohol Injection," *Arch. of Pediatrics*, Oct. 1924.

[2] L. Findlay, "Injection of Alcohol in the Treatment of Prolapse of the Rectum," *Lancet*, 1923, i. 76.

is in the outer portion of the sphincter ani. The needle then passes upward and backward towards the hollow of the sacrum, the point of the finger in the rectum guiding the needle so that it lies well outside the rectal mucous membrane. When the needle strikes the sacrum it should be withdrawn and most of the 1 c.c. of absolute alcohol injected, the remaining small portion being injected as the needle is withdrawn. The needle should be inserted on the opposite side of the anus and the same procedure followed. In this way the rectum is tacked up to the hollow of the sacrum by a small portion of scar tissue which rapidly forms. A dressing is now applied over the anus, the buttocks are strapped tightly together with adhesive and the child is kept lying flat, passing his motions also when flat. In about a week he may be allowed to run about, but he should pass his motions when lying flat for a further week or ten days. As a rule the cure is then effected.

Hernia [1]

inguinal hernia

By far the commonest form of rupture or hernia is that in the inguinal region. Here there is a continuation of the peritoneum down into the scrotum or towards the labia.

Sex-incidence.—This type of hernia is present in a proportion of 9 males to 1 female.

Clinical picture.—As a rule inguinal hernia is present at birth or shortly after birth, and is first noticed as a swelling in the scrotum, much more marked when the infant is crying. When the child is quiescent the lump seems to disappear. It may be present on one or both sides. On examination slight pressure will often replace the hernia with a gurgle, showing that it contains bowel. The author has repeatedly seen in a girl an ovary prolapsed into the sac and the cæcum and appendix prolapsed into a large hernia. The

[1] Donald Paterson and George M. Gray, "An Investigation into the Incidence of Hernia in Children," *Arch. Dis. Child.*, Vol. 2, p. 328.

size of the ring through which the hernia has passed can be ascertained by passing the little finger up the inguinal canal. Occasionally a hydrocele accompanies or is mistaken for a hernia, and thus complicates the diagnosis.

Irreducible and strangulated hernia.—Very occasionally the rupture comes down and may not be replaceable. In these cases immediate operation is called for. Should the rupture become firm and tense and threaten to become irreducible and strangulate, the following procedure should be attempted. The infant is laid on a pillow with the head considerably lower than the buttocks. A feed is given so that it may become quiet, either a bottle or the breast as the case may be. When quite quiet and sucking is proceeding, gentle pressure is exerted over the mass and an effort made to replace the rupture. With care an ice-bag may be applied. Should this method of treatment not succeed, an anæsthetic is called for, and in the relaxed condition an attempt should once more be made. Force must not be used or a rupture of the gut may result. In a very small proportion of cases operative treatment is called for.

Prognosis.—One fourth of all inguinal herniæ disappear of themselves spontaneously, during the first three years of life. The remainder will require operation sooner or later.

Treatment.—Many small illnesses are attributed to the presence of a hernia in an infant, but this is probably quite incorrect. Infants who are not treated with a truss or other appliance seem to thrive just as well as those who are treated. Nevertheless in many cases some form of retentive apparatus is indicated :

1. A woollen truss. The application of a woollen truss is not easy and the author has discarded this form.

2. A rubber horseshoe truss. This is by far the easiest to apply and the most efficient ; besides, being made of rubber, it is easily cleansed and reapplied.

Surgical treatment.—In three out of every four inguinal herniæ operative treatment should ultimately

be carried out. It is best that this operation should not take place before the age when the child is clean in its habits, that is, before the age of 18 months to two years. At this age the child is running about and there is a greater tendency, due to its activity, for the rupture to come down. The proportion of recurrences and mortality after operation is negligible.

UMBILICAL HERNIA

This is much less common than inguinal hernia and is equally common in males and females. By the age of 10 or 12 years half the cases of umbilical hernia have disappeared spontaneously, while the remainder are much reduced in size. The application of adhesive strapping over a covered-in halfpenny or a rubber truss may be recommended as treatment. In cretinism (*see* p. 333), accompanying the protruding tongue and general backwardness there is a large umbilical hernia. This answers rapidly to treatment with thyroid gland. Hypotonic, flabby, weakly children, such as mongols, tend to have widely separated recti muscles and umbilical hernia. Children with rickets and cases of cœliac disease are both prone to umbilical hernia. The treatment of the general condition of such children is essential if a spontaneous cure of the hernia is to be expected. If the rupture is of any size surgical treatment should be undertaken as soon as the child is clean in its habits.

COMBINED INGUINAL AND UMBILICAL HERNIÆ

In about one third of such cases the herniæ disappear spontaneously. The other two-thirds require surgical intervention.

CHAPTER VII

DISEASES OF THE RESPIRATORY SYSTEM

Croup

Two conditions are commonly embraced by this term :

1. Spasmodic croup or catarrhal laryngitis ;
2. Diphtheritic croup or laryngitis.

SPASMODIC CROUP

Etiology.—Spasmodic croup is due to a catarrhal infection, most commonly the Micrococcus catarrhalis, extending from the nasopharynx to the vocal cords and larynx. There is a swelling and congestion of the vocal cords which impedes their function. In addition, in children it is commonly accompanied by a greater or less degree of spasm of the laryngeal muscles.

Age-incidence.—Spasmodic croup most commonly occurs between the ages of 6 months and 3 years.

Clinical picture.—During the afternoon the child may have a slight cough and be a little feverish. After being put to bed he wakes and attracts attention by his noisy breathing. There is both an inspiratory and an expiratory stridor. Cyanosis is often present to a marked degree. All the ordinary and extraordinary muscles of respiration are over-acting. The general constitutional upset is not great, and immediately the attack is over the child seems himself again. Spasmodic croup appears therefore to consist of two separate factors, first the catarrh of the vocal cords and secondly the nervous spasm of the larynx.

The latter is the more important in producing the clinical picture.

Prognosis.—Simple spasmodic croup is seldom fatal by itself. Once an attack has occurred other attacks are to be expected.

Differential diagnosis.—It is necessary to differentiate between laryngeal diphtheria, laryngismus stridulus (a symptom of tetany or acute rickets) and croup. In *laryngeal diphtheria* the constitutional upset is severe and the colour grey. The onset is seldom sudden, often occurring in the morning or during the day. An examination of the throat as a rule shows accompanying pharyngeal diphtheria or nasal diphtheria. When there is any doubt whatever, antitoxin should be administered at once. In *laryngismus stridulus* the stridor is inspiratory and between attacks the breathing is quiescent. The face of the child with *croup* is as a rule flushed and shows the presence of some infection of the respiratory tract, a fact which is helpful in distinguishing it from diphtheria. Throat swabs should be taken in all doubtful cases.

Treatment.—Since the spasm of the larynx is much more severe during the child's waking period than during sleep, every effort to induce sleep should be made. A grain of chloral at six months of age, two grains at one year and an additional grain for each year of the child's life can be given safely three or four times daily. Where it is difficult to induce the child to swallow it, the chloral may be given rectally. Propped up in bed with a steam-kettle containing friars' balsam or a cresolene lamp, much relief is obtained. Care should be taken, however, that the steam is not on for more than 15 minutes to half an hour in three hours, as it is easy to overdo this treatment. The application of warm flannels to the neck or of antiphlogistine is recommended. The bowels should be kept well open and the diet should be light, as recommended for fever patients (*see* p. 415). Tracheotomy or intubation should not be necessary in true croup.

DIPHTHERITIC CROUP

As already stated, differentiation between this form of croup and spasmodic laryngitis is sometimes extremely difficult. Where there is any doubt whatever, antidiphtheritic serum, in doses of 4,000 to 8,000 units, should be administered at once. It may be necessary to perform tracheotomy in severe cases. Intubation, however, is to be preferred in institutions, as the sequelæ of tracheotomy are most undesirable.

Congenital Laryngeal Stridor

This is more commonly found in girls than in boys, and it is present from birth.

Clinical picture and diagnosis.—Very shortly after birth it is noted that the infant makes a noise on inspiration. It is a crowing noise and is accompanied by an indrawing of the intercostal spaces and of the episternal notch. It is worse when the child is roused than when quiescent. The physical condition of the child is as a rule excellent and the mentality normal. The voice and cry are clear and the child is normal in every way. The condition may be differentiated from that due to papilloma, where the voice is hoarse and the symptoms come on some time after birth, and from laryngismus stridulus, which accompanies acute rickets.

Etiology and pathology.—There are two theories as to causation:

1. That there is a slight malformation of the larynx, causing the vocal cords to approximate on inspiration and giving rise to the crowing and obstruction.

2. That there is no malformation, but there is a spasm of the larynx due to an inco-ordination of the respiratory muscles. It is possible that both factors operate.

Prognosis.—By the age of one year most cases have disappeared and by two years there is no further sign of the condition.

Treatment.—No treatment is of any benefit or is necessary.

Laryngeal Papilloma

This is more commonly found in girls than in boys. Whether it is present from birth is not known, but the first symptoms appear at the age of about 2 or 3 years.

Clinical picture.—The onset is very gradual, the voice being slowly lost. There is usually a huskiness only at first, then gradually complete loss of voice occurs. Later, symptoms of inspiratory and expiratory obstruction appear, which may become acute and threaten suffocation. The general health is unaffected until the later stages.

Pathology.—Growing from the vocal cords or wall of the larynx close to the vocal cords several simple papillomata are usually found. When removed they tend to recur repeatedly, but some tend on the other hand to disappear spontaneously.

Treatment.—This is largely surgical, and operative treatment should be delayed as long as possible, as the older the child the larger the larynx and the greater the opportunities of performing a successful operation. X-rays and radium treatment are claimed as successful in a minority of cases. Tracheotomy and intubation may require to be done, with operative treatment some time later.

Prognosis.—The prognosis is most unsatisfactory. Patients operated on early die of pneumonia as a rule, and when operation is left until later, such extensive removals must be done that permanent loss of voice results. Where tracheotomies are performed much trouble is given by each scar. Strenuous efforts at treatment with radium should undoubtedly be made.

Bronchitis

Etiology.—A perfectly healthy child may develop acute inflammation of the trachea and bronchi as a result of a massive infection with some virulent organism,

such as the influenza bacillus. A less virulent organism may cause this when the child has been exposed to a "chill." An ill, debilitated child may develop bronchitis, especially as an extension from an infection in the upper respiratory tract. The child with rickets and a catarrh of the nasopharynx tends to develop bronchitis by an extension of this infection. The same may be said of children with heart or kidney disease, in whom the circulation is not good. Some children develop what is termed bronchitis while they are teething, but it is a matter of doubt whether this actually is bronchitis or merely a "cough" due to an excess of saliva collecting in the region of the larynx.

Pathology. Physical signs and their interpretation.—It is necessary in the first place to define the terms to be used in describing the physical signs. The author suggests the following:

Rhonchi are dry sounds, either low- or high-pitched. There are so-called whistling, sibilant and sonorous rhonchi, and these are produced by the air whistling through the smaller bronchioles over portions of loose or tenacious adherent mucus or muco-pus.

Râles or *crepitations* (these are synonymous) may be low- or high-pitched and are short, moist sounds, small or large in size, depending on their site. The smaller ones are undoubtedly produced by the moist walls of collapsed alveoli springing apart. The larger moist sounds may be produced by air passing through mucus in some of the smaller bronchioles. Crepitations of the smaller type therefore imply lung collapse and those of the larger type imply bronchiolitis. Large moist sounds are often referred to the chest from the larynx or trachea where they are produced. This is often the case in a teething baby.

On examining the chest of an infant or young child with bronchitis the following physical signs may be found. There may be high or low whistling rhonchi present, as discerned with a stethoscope. Standing near the child he may be heard to wheeze on breathing, and if the hand is placed on the chest the same wheeze

may be detected. Again, if the smaller tubules or bronchioles are affected, and especially if the exudate is more profuse and thinner, moist sounds both small and large are present. The smaller bronchioles, becoming blocked with exudate, do not allow the air to reach the alveoli except at intervals. Minute areas of collapse are therefore present over the whole of the lung surface. With each breath some of the collapsed alveoli are inflated, giving rise to showers of fine crepitations. If low-pitched this suggests collapse only, but if high-pitched it suggests the presence of areas of consolidation or broncho-pneumonia, which convey the sounds easily to the ear. Bronchitis is, as a rule, bilateral, that is, both lungs are involved, and usually the distribution is diffuse.

Clinical picture.—It cannot be said that bronchitis has two definite consecutive stages, namely a dry stage when the surface is congested and inflamed, and a second stage when there is much mucous secretion and moisture. With some infections the bronchioles seem to stream, just as the nose does, from the beginning. With other infections there seems to be a period of dry inflammation during which the cough is short and hacking, and there is pain accompanying it, followed later by a period of moistness or looseness, when much relief is obtained from coughing up the muco-pus or bronchial exudate. The temperature is almost invariably raised in bronchitis to 100° or 101°. The type of cough depends on the nature of the secretion present. The respirations are increased in rate. The cheeks are flushed and the child is drowsy and irritable. Many children tend to be asthmatical, that is, along with an attack of simple bronchitis there tends to be an additional spasm of the bronchi and bronchioles, giving rise to so-called bronchial asthma. This is to be considered later under the heading Asthma (*see* p. 219). In such cases there are characteristic expiratory rhonchi, a feature which is not common in simple bronchitis.

Treatment. Temperature of the room.—This is best kept at about 60° F. It is necessary to have

a nursery thermometer to judge this. Warm, fresh air is absolutely essential and this may necessitate open windows. An open fire is an aid to ventilation. It is wrong to have more than one adult in the sick-room at a time.

Diet.—This should be the maximum that the child is able to digest. It is a great mistake, however, to feed small amounts often, unless food is being utterly refused. For the older child the fever diet mentioned on p. 415 should be resorted to, but the regular times of feeding should as far as possible be maintained. Between the feeds and during the night plain water, sugar-water, barley-water or diluted orange juice with sugar should be administered in large quantities. Much milk should not be given.

Local treatment.—In those cases where the cough is dry, hacking and unproductive, and an examination of the chest shows the presence of high-pitched rhonchi with a minimum of moist sounds, a steam-kettle or cresolene lamp may be indicated. The steam-kettle should not be used for longer than fifteen minutes in each three hours. A teaspoonful of friars' balsam in each kettle is useful. A steam-tent should not be used.

Drugs.—In small infants, certainly up to the age of 18 months or 2 years, cough mixtures should be avoided. Upset to the stomach is more likely to follow the use of drugs than is any theoretical benefit. In an older child two types of cough mixture may be indicated, first an expectorant mixture such as the following:

Vin. ipecac., 2½ minims
Glycer., 10 minims
Ammon. carb., ½ grain
Aq. dest. ad 1 drachm

and secondly a sedative and drying mixture such as the following:

Tr. camph. co., 2½ minims
Sp. æth. nitros., 2½ minims
Tr. bellad., 3 minims
Syrupi q.s.
Aq. anethi ad 1 drachm

The latter mixture is much to be preferred in the ordinary case of bronchitis. It is not too much to say that the use of cough mixtures is old-fashioned and inefficient. Putting drugs in the stomach to affect the bronchial mucous membrane is less likely to be successful than the method of reaching the inflamed mucous membrane by direct inhalations.

Poultices.—Prolonged or strenuous poulticing of the chest wall should not be undertaken, especially in weakly infants. A thin layer of antiphlogistine, however, across the back, and not brought forward so as to press on the walls or front of the chest, may help to relieve any tendency to spasm of the bronchioles and give comfort to the patient. The bowels should be kept well open with alkaline salts, such as Phillips's Milk of Magnesia.

As a rule bronchitis in childhood runs its course of from 3 to 10 days irrespective of any treatment other than rest in bed and careful nursing. It is unwarranted, therefore, to embark on any treatment with drugs likely to upset the infant's stomach.

Pneumonia

The two varieties of pneumonia, namely broncho- and lobar pneumonia, have a number of points in common and to a certain extent may be dealt with together.

Age-incidence.—Broncho-pneumonia is indubitably more common in infancy, while lobar pneumonia is far and away more common in the older child. Just as there is no upper limit to the age when broncho-pneumonia may be present, so there is no lower limit to the age when lobar pneumonia may be present. It seems fair, however, to say that in the first two years of life broncho-pneumonia is to be expected, and from two years onwards lobar pneumonia.

Seasonal incidence.—Cases of pneumonia are admitted to hospital most commonly in January, February and March, and again during the hot weather in July.

Before dealing with the two conditions separately we may quote the following tabulated statement of the chief differences between them[1]:—

Broncho-pneumonia	*Lobar Pneumonia*
Common during first two years of life.	Common in older children.
Subjects of weak constitution or debilitated by disease.	Subjects usually healthy and well nourished.
Due to various organisms: pneumococcus, streptococcus, influenza bacillus, etc.	Almost invariably due to the pneumococcus.
Frequently secondary to other acute illness.	Usually primary disease.
Onset slow, with symptoms of bronchial catarrh.	Onset sudden without preceding bronchial catarrh.
Temperature rising progressively and showing marked irregularity.	Temperature rising suddenly and maintaining high level.
Symptoms of pulmonary distress early.	Symptoms of pulmonary distress late or absent.
Breathing rapid, deep, laboured, costo-abdominal.	Breathing rapid, shallow, not laboured, abdominal only.
Signs of bronchitis and patchy consolidation in both lungs.	Signs of consolidation in one lobe of one lung.
Progressive signs of pulmonary embarrassment.	Progressive signs of disturbance of respiratory centre.
Cardiac trouble secondary to pulmonary lesions.	Cardiac trouble secondary to disturbance of cardiac centre.
Duration of illness indefinite; relapse common.	Duration of illness, five to ten days; relapses uncommon.
Termination of fever by lysis.	Termination by crisis.
Suppuration in other tissues rare.	Suppuration in other tissues common.
Prognosis bad.	Prognosis good.
Usual termination—death.	Usual termination—complete recovery.
Sequelæ: empyema uncommon; chronic fibroid pneumonia usually due to this disease.	Sequelæ: empyema common; fibroid pneumonia rare.

BRONCHO-PNEUMONIA

Etiology.—As a rule broncho-pneumonia may be said to be an extension from an acute bronchitis, the infection having transferred to the terminal alveoli and through the walls of the smaller bronchioles to the

[1] Garrod, Batten, Thursfield and Paterson's "Diseases of Children."

surrounding lung tissue. This type of infection is especially common following on measles and whooping-cough, both of which are accompanied by a severe catarrh of the respiratory mucous membrane. With confinement indoors in a lowered state of health, this extension of the infection to the lung substance is rapidly brought about. These so-called secondary pneumonias are very often due to the streptococcus. In other debilitating diseases, such as rickets and acute or chronic diarrhœa, pulmonary catarrh is frequently present, and in such children these infections easily extend so that broncho-pneumonia develops. So-called primary broncho-pneumonia—that is, broncho-pneumonia in an absolutely healthy child, without a prior bronchitis, is extremely rare, but it does occur. This must be due to a massive pneumonic or influenzal infection.

Pathology.—The pathological lesion in broncho-pneumonia extends from the bronchus or bronchiole in an ever-widening circle, beginning with the bronchiole itself. Supposing the lesion has been present some hours, the various areas seen under the microscope may be described in this way, commencing from the bronchiole: First there is a region where the alveoli show consolidation, some containing polymorphs, lymphocytes, endothelial cells, coagulated serum and the debris of red cells. The white cells are battling with organisms present in the coagulated serum, ingesting some of the organisms and also the coagulated serum. The next area further from the bronchiole shows the process in an earlier stage. Here the capillaries surrounding the alveoli are congested and beaded or ruptured in places and the red cells may be seen pouring out into the alveoli. Polymorphs and serum and desquamated endothelial cells are present. Still farther out the alveoli show extreme congestion in their walls only. Beyond this there is an area of collapse of the alveoli, the walls lying in close apposition. Still farther the alveoli are distended with air, and there may be well-marked emphysema present. It is suggested that this area of collapse, congestion, exudation and consolidation travels out from the bronchiole wall like the

circle of waves on a pond produced by a dropped stone. The emphysema is produced when the portion of the normal lung remaining attempts to take on the respiratory function for the damaged portion. This collapse is due to normal lung being caught between the emphysematous portion on the one hand and the damaged portion on the other, or is due to the terminal bronchioles leading to the collapsed portion being blocked with exudate. Healing and resolution must obviously occur first in the portion next the bronchioles, since this was the first portion affected, and extend outwardly.

Clinical picture. Physical signs and symptoms.—As a rule a pulmonary catarrh or bronchitis has been present for a varying length of time, possibly a few hours, days or even weeks. The extension of this catarrh to the terminal alveoli and through the walls of the bronchioles is determined by the nature of the organism and the general condition of the child. A chill, confinement, bad feeding, all lower the vitality of the child and allow this extension to take place. The temperature rises steadily from its previous height of 99° or 99.5° to 100° or 101°. The cough which has been previously present becomes much more marked and may be almost continuous. There is an expiratory grunt, and the normal rhythm of breathing is reversed, so that the pause which usually takes place after expiration now takes place after inspiration. The pulse is rapid, and instead of the normal ratio between respirations and pulse remaining as 1 : 4, it may be as 1 : 2. The rate of the respirations increases from the normal 20 or 25 per minute to 40 or even 60 per minute. The face is flushed and cyanosed, the alæ nasi work with each respiration, the diaphragm and intercostals overact. The child is restless, rolling his head from side to side on the pillow. The mouth is dry and the tongue furred. The lips are parched and the child is drowsy.

Examination of the chest.—An examination at the very commencement may show the signs of bronchitis only, harsh breathing, inspiratory rhonchi, and

occasional low-pitched moist sounds. Once the disease is established, the child's breath-sounds remain harsh, but they are higher in character. Where the small areas of broncho-pneumonia have become confluent there may be tubular or bronchial breathing. Accompanying the breath-sounds are many small moist crepitations, which are high-pitched and crackling in character, indicating the presence of areas of consolidation. The percussion note may be resonant or even hyper-resonant, due to compensatory emphysema, or may be impaired. The vocal resonance and vocal fremitus are both increased. During the course of the disease it is common for the microscopic pneumonic patches to coalesce, so that large areas, varying from the size of a shilling to a whole lobe, may give the physical signs of consolidation. In such circumstances it is difficult to differentiate between the two types of pneumonia, but as a rule pneumonic areas may be detected in other parts of the lungs.

Complications and prognosis.—Should the right heart overfill, with marked congestion and enlargement of the liver and cyanosis, the outlook is poor. This is also the case when diarrhœa accompanies pneumonia. In infants it is not uncommon and is sometimes known as symptomatic diarrhœa. Convulsions due to cerebral congestion and toxæmia are a serious complication. Abdominal distension accompanied by constipation further impedes respiration and is of frequent occurrence, requiring purges or colon lavage. Empyema is present in a proportion of cases and will be dealt with later (*see* p. 210). Another complication is bronchiectasis, which sometimes occurs, especially when the broncho-pneumonia is of streptococcal origin (*see* p. 214).

The **prognosis** depends on several factors, but probably the most important is the age of the child. The younger the child the worse the prognosis on the whole. In the first few weeks of life broncho-pneumonia is as a rule fatal. A large proportion, 80 or 90 per cent., of those who contract it up to the age of one year die. After this the mortality falls and by two years the prognosis is moderately good. If the child is under weight or

suffering from any other organic disease, the broncho-pneumonia being merely a complication, the prognosis is relatively much worse. Children with acute rickets or congenital heart disease, hare-lip or cleft-palate, kidney disease, etc., are much less able to stand pneumonia than the normal child. Accompanying or following on one of the acute infectious fevers, such as measles or whooping-cough, the prognosis is very poor. Where a large proportion of both lungs is involved, with areas of confluent broncho-pneumonia, the outlook is bad. Streptococcal pneumonia is much more often fatal than pneumococcal pneumonia.

Course of the disease.—This type of pneumonia lasts from one to three weeks. The onset is gradual and the cessation is by lysis (*see* Fig. 27). There is a tendency to fresh patches and recurrences.

Treatment. *Position.*—The patient

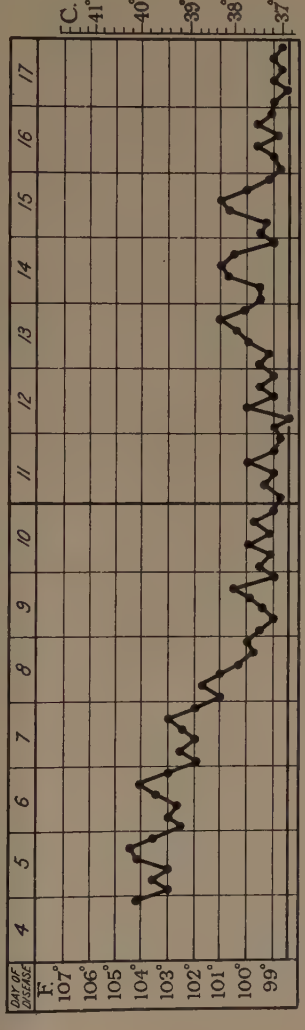

Fig. 27.—Chart of a child with broncho-pneumonia, showing temperature falling by lysis.

should be kept in bed and propped up in a reclining position. No drug or other form of treatment has any definite effect on pneumonia, and we should therefore aim at conserving the strength of the child.

Clothing.—Light, loose clothing of wool or silk and wool should be worn, or a light gamgee pneumonia jacket. The bedclothes should be light and may be held off the lower limbs of the child with a cradle when the temperature is especially high or if the weather is warm.

Fig. 28.—Method of giving intranasal oxygen in pneumonia.

Fresh air.—Since the aerating surface of the lungs has been greatly diminished, as much oxygen as possible should be present in the air to enable the lungs to perform their function. Often it occurs that when an infant or child is ill, the whole anxious family gather in one small room and use up the available oxygen, thereby harming the child. Should such be the case, a cylinder of oxygen, provided with a length of rubber tubing and a funnel, should be obtained and the oxygen administered for 5 or 10 minutes in each hour. A much better plan is to give the oxygen by means of a nasal tube, bubbling from a Woulfe's bottle. In this way it can be given

continuously and the whole of the oxygen is obtained by the child. If the child is restless cardboard splints can be placed on the arms (Fig. 28). If, however, the room is large and the air is cool this is not necessary, unless the case is extremely severe. Oxygen therapy should be begun much earlier than formerly was the case, and one should not wait until the child is almost moribund.

Stimulants.—In infants it is seldom that death takes place from cardiac dilatation, but in older children this may play a great part. Where there is anything more than a very slight involvement of the lungs tincture of digitalis should be given. The dose for a child of $1\frac{1}{2}$ years would be 6 drops in 24 hours. Brandy, caffeine, camphor, strychnine, and other stimulants are only advisable as a last resource. It is seldom that they are really useful or necessary, and hypodermic injections as a routine should be discouraged. If the chest is moist, the whole lung tissue being filled with fluid, atropine or belladonna is indicated. Atropinæ sulphas, $\frac{1}{200}$ to $\frac{1}{100}$ gr., may be given hypodermically on two or three occasions, just to dry up the chest and thus extend the pulmonary ventilating surface.

Diet.—This should be light, not more than half that given in health, and accompanied by quantities of bland fluids. For a child of a year, half a pint of skimmed milk spread over the day is sufficient. For breakfast, Groats, Benger's, Savory and Moore's, or some other starchy or cereal food should be given; for dinner, broth or soup, thickened with a little potato; for tea, give much the same food as at breakfast. During the night, if the child is awake, a further starchy milk feed may be given. It is well to keep the fat low, much milk and eggs being contra-indicated. Quantities of orange-, lemon- or grape-water, well sweetened with sugar, and barley-water should be given between meals. If this régime be adhered to, the child will be less inclined to refuse food and will show a cleaner tongue. In addition there will be much less likelihood of his developing an accompanying acidosis.

Sleep.—Perfect quiet should be maintained and every effort made to get the child to sleep as much as possible. Taking the temperature, changing and general attention should be done at as long intervals as possible, so as to ensure the maximum of sleep.

Poultices.—The poulticing of the chest in infants or weakly young children must be done with great care. A heavy poultice applied to the chest of a child whose strength is already failing may just make the difference between recovery and death. The added weight on the chest and the impediment to its free movement often does much more harm than good. As far as possible nothing should be placed on the front of the chest. Probably one of the best fomentations is antiphlogistine, which may be applied with benefit across the back, being brought forward a little into the axillary region. Great care should be taken to keep the skin well oiled with olive oil, whatever form of poultice is applied. In the vast majority of cases poulticing is not indicated.

Leeching.—In cases of capillary bronchitis with broncho-pneumonia where from the very start there is cyanosis and embarrassment of the right heart, a leech applied over some bony prominence is of value. The removal of some of the excess of blood seems to remove the embarrassment of the right heart, for the time being at least.

Steam tent.—This is probably one of the most dangerous of the old methods of treatment and to-day it is not used, except with extreme care. It is difficult to combine fresh air and steam, and there can be no question that fresh air is an absolute necessity. When steam is introduced into the room, it should never be for more than fifteen minutes in every three hours. A steam tent should not be utilized at all in broncho-pneumonia.

Tepid sponging.—During the hyperpyrexia sponging the face and limbs with tepid water is indicated. The temperature should not be allowed to rise above 104½° or 105°, because of the tendency to convulsions.

LOBAR PNEUMONIA

Etiology.—The etiology of lobar pneumonia is still obscure. Since the onset is abrupt and accompanied by rigors, and since organisms have been cultivated from the blood by various workers, it may be assumed that it commences as a pneumococcal septicæmia, one or more lobes being involved in all its parts.

Pathology.—There are four stages :

1. The stage of congestion, where the capillaries surrounding the alveoli are distended and appear beaded under the microscope.

2. The stage of red hepatization, where there is an exudate into the alveoli containing blood, serum, polymorphonuclear leucocytes and desquamated endothelial cells.

3. The stage of grey hepatization, where the exudate into the alveoli shows coagulation

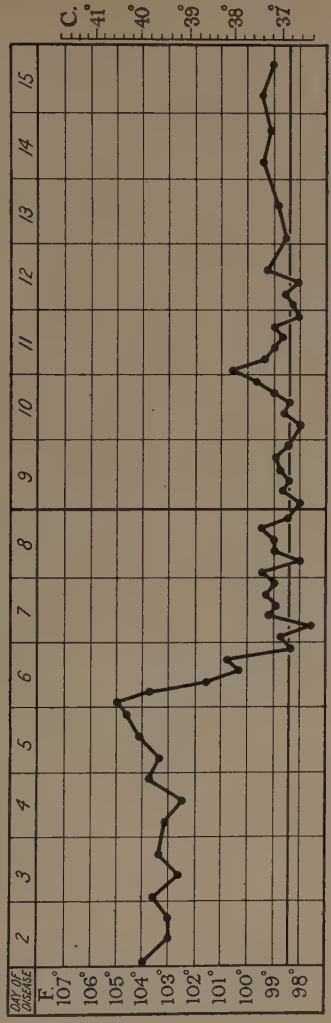

Fig. 29.—Chart of a child with lobar pneumonia, showing temperature falling by crisis.

and the red cells, which have burst into the alveoli are devoured and decolorized by the white cells present. The alveoli are less distended and there is a tendency for liquefaction of their contents.

4. The stage of resolution. This stage consists in the liquefaction of the alveolar contents, the debris or pus being coughed up. During this process the lung gradually assumes its normal functions. This stage may be delayed for days or even weeks (Plates 5-7).

Clinical picture: Physical signs and symptoms. —A typical description of lobar pneumonia is as follows : A child, say aged five, has been to some sports and become over-heated or wet with rain and chilled on the bus returning. That evening he vomits and there may be a definite rigor, he is flushed, his temperature rises to 104° or 105°, the respirations are rapid, 35 to 40, and the pulse rises to say 120 or 130 and is full and bounding. The following day the picture is much the same, but in addition the respirations are grunting and the respiratory rhythm is reversed, the tongue is dirty and there is a disinclination for food. The child by now has a short, irritating cough, which continues at short intervals. It is often accompanied by a stabbing pain on the affected side. After six or eight days of this, during the latter part of which the colour may become slightly cyanosed and the child a little light-headed or delirious, the temperature suddenly settles to normal (Fig. 29) and quite quickly, in three or four days, he seems himself again. Accompanying the pneumonia there is often herpes about the mouth.

The onset may be accompanied by convulsions, in fact in young rachitic children this is to be expected. In examining the child extremely early in the disease there may be no physical signs whatever. Within 24 or 48 hours, however, the breath-sounds over the affected lobe will first be noted to be diminished, so that the air entry is poor, and the normal harsh breath-sound may appear exaggerated at the other base. As a rule the base where the breath-sounds are damped down or diminished is the affected one. Accompanying this

Plates 5, 6, 7.—Radiograms showing the resolution of a case of lobar pneumonia over a period of one week.

Plates 5, 6, 7.—Radiograms showing the resolution
of a case of lobar pneumonia over
a period of one week.

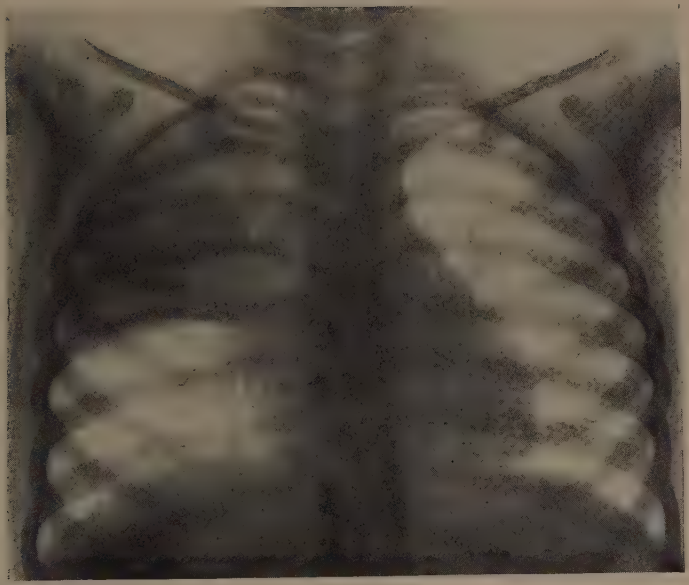

Plate 5.—Radiogram taken after the crisis.

period of diminished air entry and breath-sounds there is slight impairment of the percussion note. A few hours later, on listening over the affected area, tubular or bronchial breathing may be heard unaccompanied by added sounds. Both the vocal fremitus and the vocal resonance are increased. The percussion note is grossly impaired and is dull, but not stony dull as when percussing over fluid. Very often when listening at the opposite base bronchial breathing, slightly less intense, may be heard here also. This is as a rule merely referred from the opposite side. On the 7th, 8th, or 9th day, accompanying resolution, the physical signs change. Accompanying the bronchial breathing there are high, fine, crackling crepitations. A little later the accompanying sounds are larger, more bubbling, as if produced in the smaller bronchioles, and lower in pitch, showing less consolidated lung to convey them to the listener's ear. Finally tubular breathing clears up and the moist sounds also, leaving a normal, healthy lung. These signs are shown during the stage of resolution and the crepitations are known as "redux crepitations."

Different types of pneumonia.—There is what is known as the abortive type of pneumonia, where on the 2nd, 3rd, or 4th day all the physical signs suddenly clear up. On the other hand, in a certain proportion of cases the pneumonia continues for a fortnight or even three weeks. In certain cases no physical signs are present until just about the crisis or even after the crisis has occurred. In apical pneumonia the symptoms are very often largely cerebral and what is called meningism is seen, that is the patient is delirious, holds the neck stiff, may squint, and looks in fact quite like a case of meningitis. These symptoms clear up when the physical signs become established.

Complications.—By far the commonest complication is the development of pleurisy with empyema. Pneumococcal pericarditis does occur but is not common. This condition is dealt with elsewhere. Pneumococcal arthritis, peritonitis, meningitis, secondary to pneumonia, are also extremely uncommon. Occasionally cerebral

thrombosis has been recorded. Sometimes the pneumonia fails to resolve, and this may be because of the type of organism. Enlarged glands pressing on the communicating bronchioles may prevent the pus from draining away. In either case there is a tendency for chronic fibroid lung with bronchiectasis to follow as a complication.

Prognosis.—On the whole the prognosis in lobar pneumonia is excellent, the mortality being only about 4 per cent. in all cases, and much smaller in patients over 2 years old. In no disease in childhood does the patient appear so ill, and yet so seldom die. When he seems as bad as possible he suddenly recovers in a dramatic fashion. Complications such as empyema, fibroid lung and pericarditis affect both the immediate and the ultimate prognosis for the worse.

Treatment.—Since the mortality is extremely low in lobar pneumonia and if left, given careful nursing, almost all uncomplicated cases over the age of two recover, there is little call for heroic measures in our present state of knowledge. Fresh air, careful feeding, occasional oxygen, supervision of the child's sleep, tepid sponging when necessary, and careful watching for complications are all that is required. For details of the treatment see Broncho-pneumonia (pp. 204-206).

Empyema

This is a purulent effusion into the pleural cavity, as a rule following pneumonia.

Etiology.—In the vast proportion of cases the organism present in both the primary pneumonia and the secondary empyema is the pneumococcus, but in a certain number of cases the streptococcus, staphylococcus influenzal bacillus and tubercle bacillus may be found. It is possible that, accompanying the primary pneumococcal septicæmia, some of the organisms settle in the lung and give rise to lobar pneumonia, while coincident with this there is a purulent pleurisy. In some cases, however, it seems clear that there has been an extension

from the lung to the pleura at an interval of some days after the actual onset of the pneumonia.

Pathology.—The pleura being affected, the thin almost colourless effusion rapidly fills the pleural cavity. Quickly this becomes milky, opaque, and later purulent. Within a few hours a thick layer of felted lymph coats the parietal and visceral pleuræ. If this is opened and drained large clotted lumps are evacuated. There is a tendency for the pleura, whether drained or not, to be permanently thickened over the surface of the lung, and this thickening tends to extend into the lung substance, giving rise to interstitial pneumonia. Often the two layers of pleura are bound together after an empyema, or strong adhesions are formed.

Clinical picture.—There are two common types of empyema. In both, the clinical picture is much the same. In the first, which is much the commoner, the child has what appears to be an ordinary pneumonia of the lobar type. The temperature settles on the 7th or 8th day and he appears to be doing quite well. Three or four days later the temperature rises slightly and the child does not seem so well. Each day the temperature is slightly higher, until it may reach 102° or even 103°. On investigation the pleural cavity is found to contain pus. The other type is less common, and in this the temperature shows no inclination to settle down at the end of a week or 10 days, although an ordinary pneumonia has been diagnosed. After having continued high for some days or longer, and the physical signs becoming more those of fluid than pneumonia, an investigation is made and pus is found. In the first type it is probable that the pleural cavity was infected secondarily to the lung, whereas in the second type it is considered probable that the pleural cavity and the lung condition were coincident and part of a general pneumococcal infection. The prognosis in these two types is quite different and they must be considered from time to time separately.

Among the symptoms are pain in the affected part of the chest, a short hacking, unproductive cough,

dyspnœa getting more and more marked, and cyanosis also progressively more marked.

On examination, the breathing and pulse-rate are noted to be extremely rapid. There is displacement of the heart, the direction depending on which side the empyema lies. The child is cyanosed and the heart is displaced to the side opposite to that containing the empyema. On placing the hands over the two sides of the chest a definite thickening and œdema of the chest-wall is to be made out on the affected side. On percussion the note is stone-dull, either over the whole lung or over the lower part, where the fluid has collected. On auscultation the breath-sounds are either absent or much reduced. At the upper level of the fluid there is ægophony. Vocal resonance and vocal fremitus are decreased. On palpation of the abdomen, if the heart has been embarrassed the liver will be found to be enlarged.

Course of the disease.—Should the pus not be evacuated, the fever continues for some weeks or months. The pus may gradually point at or somewhere about the nipple or in the axilla, becoming a so-called "empyema necessitas." On the other hand, in pure pneumococcal infections the pus may be gradually reabsorbed, leaving greatly thickened and adherent pleuræ. It is in this type that there is a tendency for interstitial pneumonia to develop, giving rise finally to a fibroid lung with bronchiectasis. This type is, however, uncommon.

Prognosis.—The prognosis very largely depends on :

1. The age of the child. As in broncho-pneumonia, in the very young child the prognosis is extremely bad. Under one year there is a mortality of at least 75 per cent., but this diminishes rapidly up to the age of two, when the mortality is comparatively small.

2. The previous health and general physical condition of the child. In rachitic or under-nourished children, or those children who have some organic disease elsewhere, the chances of recovery are greatly lessened.

3. The nature of the organism. In pneumococcal

empyemata the prognosis is best, whereas in streptococcal or influenzal empyemata the outlook is much graver.

Treatment.—Even if pus is suspected and found during a pneumonia, thorough drainage is still undesirable. A needle may be put in from time to time and the greater part can be evacuated by means of a syringe, but the pneumonia should be allowed to clear up, and the pus then dealt with by resection of a rib or the introduction of a tube between the ribs. Where the empyema is merely part of a general septicæmia, it is much the best course to wait until it shows signs of loculation or subsidence, and it may be necessary to wait some days before this occurs. The introduction of a needle and the aspiration off of pus from time to time is called for if there is cardiac embarrassment. If pus is definitely detected and a decision has been made to evacuate it, resection of a rib or intercostal drainage is much to be preferred. When a rib has been resected and an extensive opening made in the chest-wall, a finger may be inserted, breaking down adhesion between the layers of pleura and evacuating large clotted lumps. This tends to prevent permanent adhesions, thickening of the pleuræ and interstitial pneumonia. The tube should be left for some days until no discharge appears, as there is a great tendency to too early closure. Intercostal drainage by the de Pezzer self-retaining drainage tube, combined with washing out of the pleura daily with some mild antiseptic such as Eusol or Dakin's solution, has been successfully carried out of recent years in hospital practice. In this way osteomyelitis of the ends of the ribs, so prone to occur after rib resection, is avoided. Once the pleural cavity has been closed, such exercises as blowing into a Woulfe's bottle, inflating toy balloons, blowing on whistles and horns, etc., which tend to cause full expansion of the lung, are to be encouraged. Sea-air shortens convalescence and improves the child's general health. Cod-liver oil is indicated in this condition.

Complications.—Accompanying an empyema and producing a fatal result have been found the following:

pyopericardium, suppurative meningitis, lateral-sinus thrombosis and gangrene of the lung. After operation in some cases there is still a high temperature and persistent toxæmia. Late in the course of the disease severe diarrhœa is often a complication. In such cases there is probably an accompanying septicæmia, and this is as a rule fatal. The author has found blood transfusion most useful in these circumstances.

Bronchiectasis (Bronchiolectasis—Fibroid Lung—Interstitial Pneumonia)

Etiology.—Fibroid lung, or bronchiectasis, is most commonly the sequel of unresolved pneumonia, the pneumonia being as a rule of the broncho-pneumonic type, following on measles and whooping-cough. The streptococcus is therefore most commonly the organism concerned. In little children the tubercle bacillus is seldom the primary cause of the condition, but later it may attack the damaged lung. Fibrosis of the lung, arising from the pleural surface and passing inward by the septa, may occur following on an undetected empyema. Foreign bodies in the bronchus and enlarged glands pressing on the bronchioles may cause retained pulmonary secretions with a low-grade pneumonia and finally bronchiectasis.

Pathology.—With the ordinary pneumococcal infection the interstitial tissue of the lung is less affected, the brunt of the infection falling on the alveoli themselves. In streptococcal infections the inflammatory reaction in the interstitial tissue is great, with consequent scar-tissue formation and contractures. If the inflammation is slow and long-continued, fibrous tissue is invariably formed, with permanent damage and scar-tissue formation. The tubercle bacillus, the spirochæte, or some less virulent and lower-grade organisms may also cause fibrous-tissue formation (Fig. 30).

Symptoms and diagnosis.—There may be a complaint that, following on a broncho-pneumonia after measles or whooping-cough, the child has never been

well since. An examination will show bronchiectasis. On the other hand, in some cases no history is obtainable to help in this matter, the process having apparently come on slowly and insidiously. A severe *cough* is always a feature. It may be barking and harsh, or brassy, suggesting enlarged mediastinal glands or a foreign body. Often it is accompanied by an inspiratory whoop, very suggestive of whooping-cough. The breathing is more

Fig. 30.—State of lung in bronchiectasis.

rapid than normal, and on exertion the child may become cyanosed and dyspnœic. There may be *clubbing of the fingers* in cases of long standing. Probably one of the first things to be noticed is the *odour of the breath*, which becomes fœtid and offensive. The *sputum* is characteristic, containing much pus, and may amount to an ounce or more in the day. The paroxysms of coughing are not frequent, perhaps three or four times in 24 hours, but as a rule these are productive, if the child is in the proper position. On *examining the chest* there is often definite flattening of the chest or retraction of the ribs on the

affected side. The left lower lobe is much more commonly affected than the right; possibly there is some connexion between the pressure of the heart on the left bronchus and bronchiectasis. On *percussion* the note is dull over the affected lobe. It may be so dull as to suggest fluid, but it is usually what may be termed apple dullness, not stony dullness. On *auscultation* the physical signs vary enormously, depending on whether or not the tubes contain fluid. If they are well cleared out the breath-sounds are tubular or amphoric, suggesting cavity-formation. In addition the accompanying sounds are high-pitched, large and splashing. If the tubules are partially filled the breath-sounds may be markedly diminished and distant, with a few accompanying moist sounds only. If quite filled, as they are at certain times in the day, the breath-sounds may be quite absent and there may be no added sounds whatever. By inverting the child, so that the secretions tend to drain away, coughing is provoked and the cavities emptied, and in a few moments the entire auscultatory signs are changed. The question whether fluid is present in the pleura will often arise. It should be noted that in bronchiectasis the heart is drawn over to the affected side, and not pushed away from it as with fluid in the pleural cavity. The chest-wall is definitely flattened on the affected side also. An X-ray of the chest shows a displacement of the trachea and other contents of the mediastinum to the affected side. The affected lobe of the lung casts a shadow, which may in some cases be definite enough to suggest empyema.

The question continually arises: is this pulmonary lesion tuberculous or non-tuberculous? A careful examination of the sputum will help, but on the other hand it should be realized that a non-tuberculous fibrosis may at any time become infected with the tubercle bacillus. In children a tuberculous pulmonary fibrosis tends to be progressive or to become miliary and end fatally.

Lipiodol.—The modern investigation of bronchiectatic cavities by means of lipiodol has thrown much

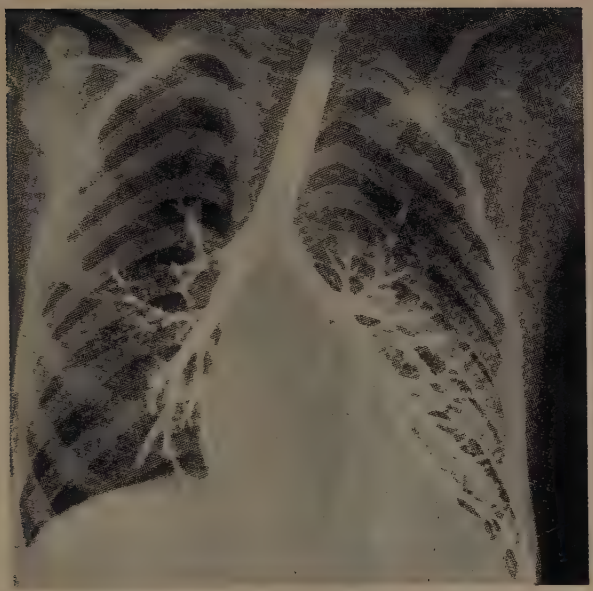

Fig. 1.—Normal bronchial tree in a case of slowly resolving pneumonia.

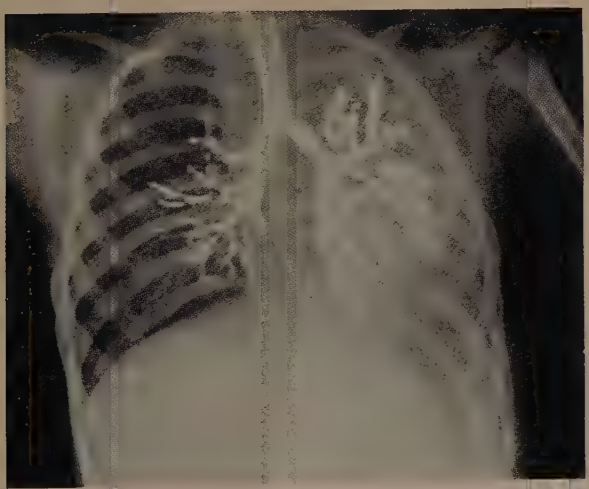

Fig. 2.—Extreme degree of tubular bronchiectasis throughout whole of left lung.

(By courtesy of Dr. Leonard Findlay.)

Radiograms taken after lipiodol injection.

Plate 8.

light on this whole condition. The procedure, however, is not without danger and should on no account be undertaken by anyone who is not skilled in its technique (Plate 8).

Prognosis.—A slight pulmonary fibrosis is quite compatible with good health and normal longevity; in fact, after a few months of treatment, no physical signs are given, nor can a pulmonary lesion be detected by X-rays, the chest appearing to be quite normal. If, however, the child's health falls below par for any reason, recurrent bronchitis or pneumonia is prone to occur, and the fibrosis may progress to bronchiolectasis or bronchiectasis. In time, in the severe cases, cardiac failure may occur, with marked clubbing of the fingers, increasing cyanosis and dilatation of the heart. Much displacement of the heart, due to adhesions in the pleura dragging over the mediastinal contents, may largely account for this. Occasionally in the most severe cases, where the sputum is fœtid and offensive, cerebral abscess occurs. In some cases hæmoptysis, especially when there is large cavity-formation, may prove to be a terminal event.

Treatment.—Each case should be treated on lines similar to the modern method of treating pulmonary tuberculosis. Fresh air is essential. An open-air life should be arranged and continued for long periods. The diet should be well-balanced and nourishing, and cod-liver oil is indicated. The posture of the patient, with efforts at coughing to empty the cavities two or three times a day, is a most important adjunct. Even a very small patient can be taught to do this perfectly. A mixture containing:

Tr. benzoin. co., 2½ minims
Tr. camph. co., 5 minims
Creosote, ¼ minim
Syrup of tolu, 5 minims
Mucilage of acacia, 10 minims
Peppermint water to 1 drachm

(the Hospital for Sick Children mist. benzoini co.), is of benefit. The best medicinal treatment is inhalation with a Burney-Yeo mask; 2 or 3 drops of a mixture of

creosote, 1 part, and chloroform, 2 parts, should be given at intervals of three or four hours.

Surgical treatment.—The treatment of pulmonary fibrosis in childhood surgically has not been sufficiently attempted. In adults the removal of large portions of the ribs on the affected side, allowing a massive collapse of the damaged lung, has been accomplished successfully in a great many cases, apparently with perfect results, the sputum drying up and the cavities being obliterated. In a certain number of children this procedure has also attained success, but further experience of it is required.

Enlarged Thymus

It is seldom that simple idiopathic enlargement of the thymus in young infants gives rise to symptoms of respiratory obstruction. The author has never seen such a case. In cases of sudden death (status lymphaticus), however, enlarged thymus has been found with enlargement of the lymphoid tissue throughout the body. In such cases it is the enlarged thymus which has been blamed for the sudden death. Where there is a family history of this, an X-ray examination of the chest should be taken, and if any undue enlargement of the thymus is detected, an exposure or two to X-rays will reduce this.

In so-called *thymic asthma* in infants there is very often a gross enlargement of the thymus due to some inflammatory process. Much the commonest is a tuberculous infection of the thymus with caseous masses present in it or even a cold abscess. Such cases are not uncommon and have been proved post mortem. The author has seen one case in an older child where there were constant dyspnœic attacks early in the morning, and on X-ray of the chest a much-enlarged thymus could be seen. With repeated deep X-ray therapy and a diminution in the size of the thymus, the attacks ceased.

Plate 9 shows the X-ray of an infant aged 10 weeks, the child of a tuberculous parent, who developed symptoms of obstructed breathing and physical signs like those of asthma, dying a few weeks later. At

Plate 9.—Radiogram showing enlarged thymus in an infant ten weeks old.

post-mortem a massive tuberculous infection of the thymus was found.

Asthma

Etiology. **Age.**—Asthma may occur at any age and some cases appear to commence almost from birth. Two-thirds of the cases occurring before puberty commence before the age of 3 years.

Sex.—There would seem to be a proportion of at least two boys to one girl.

Heredity [1].—In more than half the cases there is a family history of asthma, hay-fever, eczema or urticaria. Migraine, train and bus sickness have also a familial tendency. This would suggest a marked tendency for asthma to be inherited, or at least for an instability of the nerve supply of the bronchioles to be hereditary.

Exciting factors.—There are various factors which may precipitate an attack of asthma. Among the commonest are (1) infections, especially of the naso-pharynx, (2) digestive upsets and bilious attacks, (3) over-excitement and over-exertion, (4) hypersensitiveness to some protein, such as egg, feathers, fur, nuts, etc.

Climate.—Undoubtedly climate plays a great part in the production of asthma. For instance, on a sultry July day a number of patients will present themselves at the out-patient clinic with an attack. There may have been a period of cool weather, suddenly followed by warm weather, when the children are both over-clothed and overfed. Again, an epidemic of colds or influenza may have its crop of asthmatics. It is claimed by many that an east wind produces asthma, the reason for this not being clear.

Whether an asthmatic child may suffer at one time from attacks due to infections and at another time from attacks due to dietetic indiscretions, over-excitement and over-exertion, or to anaphylaxis to some foreign protein, is not certain, but undoubtedly more than one factor may operate in an individual case. A change to the

[1] G. W. Bray, "The Hereditary Factor in Asthma and Other Allergies," *Brit. Med. Journ.*, Mar. 1, 1930.

seaside may for the time being relieve the child of all symptoms, but on his returning home they may tend to recur. In the British Isles the Isle of Wight is the favourite resort for asthmatics.

Allergy (anaphylaxis to protein).—In a certain proportion of cases asthma is undoubtedly due to a pathological reaction of the body to some animal, vegetable or bacterial protein. By careful skin-tests the offending protein may be determined, and with its withdrawal there is a cessation of the symptoms.

There can be no doubt that some other missing factor must account for the large proportion of cases which cannot be put down to allergy. Cases occur more commonly at week-ends than during the week, and they occur at night more commonly than during the day. An attack of influenza passing round a family precipitates asthma in our patient. Over-eating on eggs and cream produces biliousness in some children, but asthma in our patient. Over-exertion and excitement produce irritability and bad temper in the ordinary child, but are followed by an attack of asthma in the asthmatic. What is the missing factor?

Clinical picture.—An attack of acute spasmodic asthma may be ushered in with a digestive upset or sneezing. In about half the cases there is a moderate degree of fever. The length of the attack depends on the type of asthma, varying from a few hours to a week or more. Some children have a mild or severe attack every night. The child may be put to bed with what appears to be a slight cold, but wakes during the night much distressed, with coughing and wheezing, and is unable to get his breath. He is noticed to be cyanosed, his respirations are rapid, and he looks anxious and careworn. While asleep the attack is much less severe and may even pass off, but while awake all his symptoms are augmented. Those cases accompanied by fever are difficult to differentiate from a bronchitis or a pneumonia, especially in infants. When the acute spasmodic condition passes off, generalized true bronchitis often remains.

Classification.—The term *spasmodic asthma* may be reserved for those cases starting very acutely and accompanied by extreme cyanosis and respiratory distress. In *bronchial asthma*, on the other hand, the spasm is less marked, with less distress, but as a rule there is an accompanying or superadded bronchitis, with a mild degree of fever. The author believes that there is no clear-cut type of asthma, the attacks being sometimes short and sharp and sometimes long-drawn-out, lasting several days. The individual does not consistently suffer from the same type of attack, and is sometimes febrile and at other times afebrile.

Physical signs.—The percussion-note is unchanged or hyper-resonant. On auscultation both inspiratory and expiratory rhonchi are to be heard. The excursion of the chest is small and the air entry extremely poor. The chest is barrel-shaped.

Diagnosis.—In every case organic pulmonary disease must be carefully excluded, as asthma may be simulated by such conditions as fibroid lung or bronchiectasis, especially cases which are of long standing. An X-ray of the chest should be undertaken whenever possible, and an intracutaneous tuberculin (Mantoux) test is of value as a routine. Repeated attacks of bronchitis for no apparent cause in a child should always suggest the possibility of asthma. The blood-count showing an eosinophilia of from 4 per cent. to 10 per cent. or even 20 per cent. is most suggestive of asthma; so also would be a strong family history of this disease. Asthma should also be suspected if the child has a previous history of well-marked eczema or urticaria.

Preventive treatment.—In those cases due to repeated *infections* a search must be made for the septic focus, and septic teeth or tonsils removed. In this group may be found children with small inadequate noses, and these cases do extremely badly until puberty, when the nose enlarges and the asthma ceases.

The *dietetic régime* of the child should be carefully considered. Eggs, fish and milk or cream must be examined in relation to the attacks. The bilious attack

producing a metabolic upset may bring on asthma, and the prevention of this may save an attack. The exclusion of certain articles from the diet may prevent asthma by removing the exciting factor. By the protein skin-tests the offending substance should be readily ascertained.

Over-excitement and over-exertion must be avoided. It is interesting to note how rapidly the symptoms of the asthmatic child clear up when he is removed to hospital from home. The additional rest obtained in the ward is responsible for this. Children out of school, during Saturday and Sunday, tend to be on their legs too much, when they become over-tired and get attacks. In the same way they are also too tired in the evenings, when attacks commonly occur.

Undoubtedly $\frac{1}{4}$ grain of Ephedrine (Burroughs, Wellcome & Co.), administered night and morning, week in and week out, helps to ward off attacks, and gives a child a rest and a chance to improve in health. A very useful mixture, which acts both as a tonic and as an antispasmodic and expectorant (chiefly, of course, when given as a routine) is :

Liq. arsenicalis, 1 minim
Pot. iod., 2 grains
Tr. belladonnæ, 3 minims
Aq. chlorof. ad 1 drachm t. d. s.

For those cases which are thought to be due to anaphylaxis to some absorbed foreign protein that is being ingested, the following mixture is found of great help :

Liq. adrenalin hydrochlor. (1 in 1,000), 5 minims
Acid. hydrochlor. dil., 30 minims
Pulv. dextrose, 30 grains
Syr. senn., 20 minims
Aq. chlorof. ad 2 drachms

To be taken in a cup of water with meals.

Treatment of an attack.—If an attack is anticipated the child should be given a preliminary purge and a suitable dose of Dover's powder to ensure a good night's rest. For a child aged two, give half a grain, and repeat this in an hour if he is not sleeping well. For a child of seven years give 2 grains, with a further 2 grains in an

hour if necessary. A grain of chloral per year of the child's life is also suitable, and can be repeated at two-hourly intervals until it is effective. Immediately the attack starts 1 to 2 minims of a solution of 1-in-1,000 adrenalin should be given subcutaneously, or Ephedrine (Burroughs, Wellcome & Co.), $\frac{1}{4}$ grain, should be given by the mouth. In a certain proportion of cases the attack is promptly aborted by the adrenalin. With the Ephedrine the action is much slower. The use of a cresolene lamp is advisable only if there is much accompanying bronchitis. For the costive bowels a sharp purge may be given, either 3 to 5 grains of grey powder or half a grain of calomel. This should be followed the next morning by milk of magnesia or Eno's Fruit Salts.

Diet.—No eggs or cream should be given during the attack, and all milk should be well skimmed. Horlick's Malted Milk is useful. For children of three and upwards weak tea, well sweetened with sugar, makes a good substitute for milk. Veal- or beef-broth, thickened a little with potato, should be given at midday and the child should be kept under- rather than overfed until his symptoms clear up. A plentiful supply of carbohydrate, either as plain sugar or barley-sugar, is useful, and this does away with any accompanying acidosis.

Skin protein tests.—To investigate a case of asthma the child's *home environment* must first be examined, and such factors as a feather-bed, dogs, cats, chickens or horses in close proximity, factory odours and growing plants should be inquired into.

Skin-tests are best done with solutions prepared from the child's own surroundings, e.g. his feather-pillow or bed, horsehair mattress, cat- or dog-hair. Next *dietetic* factors, such as meat, fish, eggs, milk, vegetables, fruit, nuts and seasonings should be tried in the same way. The common *bacteria* should also be excluded as causal agents.

Technique of performing skin-tests.—Although the stock dried powders and solutions which can be purchased are worth trying, it must be admitted that to do these tests satisfactorily fresh preparations are absolutely necessary, and this entails a special apparatus

and a special knowledge which is not available to the average general practitioner.

The child's arm is cleansed and a series of small scarifications are made without drawing blood. The fresh substances, dried protein powders, pastes or solutions are used, the fresh substances being the most serviceable. Each scarification is softened with a drop of decinormal sodium hydroxide solution, and the substance carefully rubbed in. Positive reactions commence within 20 minutes and fade in about an hour, a localized wheal with a surrounding erythema showing in a positive case. Delayed reactions may occasionally appear at the end of 5 or 24 hours.

Where a positive reaction is given the offending substance is either (1) removed, or (2) given in minute doses by the mouth, or (3) the child is desensitized by hypodermic injections. These methods constitute the specific treatment. Non-specific treatment is described in the next paragraphs.

Peptone.—In a small series of cases admitted to hospital for intravenous injection of peptone, a certain amount of success might be said to have been achieved. Starting with 1 minim of a solution of Armour's peptone, an additional minim was given each third day until 10 minims had been reached. As the result of protein-shock the patients appeared to be much improved and for many months afterwards were quite free. They gradually resumed their asthmatic tendencies, however.

It has repeatedly been noted that after a severe pneumonia, measles or whooping-cough infection, many months may elapse before there is a recurrence of the asthma.

Tuberculin.—The author has no knowledge of this treatment subcutaneously, but it has been claimed that small subcutaneous injections have excellent therapeutic results.

Prognosis.—The vast majority of cases of asthma occurring in childhood cease before puberty. There are likely to be permanent emphysematous changes with deformity of the chest in a proportion of cases.

Pulmonary tuberculosis.—For tuberculosis of the respiratory system, *see* Chap. XVII, p. 417.

CHAPTER VIII

DISEASES OF THE CIRCULATORY SYSTEM

Heart Disease

A good average for the rate of the heart in childhood is given by Holt and is as follows:

Pulse-rate.		*Respiration-rate.*	
At birth	140–120	At birth	50–32
6 to 12 months	115–105	First year	35–25
2 to 6 years	105– 90	2 to 4 years	25
11 to 14 years	85– 75	5 to 14 years	25–20

Increased heart's action (tachycardia)

Nervous tachycardia.—The rate of the heart's action varies with the age and temperament of the child. It also varies with the time of day, being much faster during the day-time than when the child is asleep. To get a true picture of the rapidity of the heart the rate should be taken after the child goes to sleep at night and before he wakes in the morning, as it is inclined to be 20 or 30 beats above the normal, even when resting, if he is awake. In disease, however, this variation between the waking and sleeping pulse-rate is much less. For instance, in pneumonia and other acute infections there is no difference at all. The wider the variation between the night and day pulse-rate the less likelihood there is of organic disease of the heart.[1] In pink disease (*see* p. 310) and in miliary tuberculosis (*see* p. 423) the cardiac rate is extremely rapid and should arouse suspicion at once. Fear, apprehension and excitement will raise the pulse-rate of some children many beats for long periods. This should not be forgotten.

[1] G. A. Sutherland and John McMichael, "The Pulse-rate and Range in Health and Disease during Childhood," *Quart. Journ. Med.*, Vol. 22, No. 87, p. 519.

Rheumatic tachycardia.—In most cases of acute or chronic rheumatism the heart's action is increased. This is not invariably so, but can be taken as a general rule. With each fresh outburst of rheumatism the rate is increased, and as the infection subsides, so the rate subsides. Until perfect compensation has been established, however, the heart-rate is inclined to be above normal, although quite steady. In contrast to the nervous increase, the rheumatic heart does not return to the normal rate during sleep.

True paroxysmal tachycardia.—This is very rare in infancy and childhood, but there are a few authentic cases on record, which are associated as a rule with congenital heart disease. A gross organic lesion in the heart-wall, encroaching on the sino-auricular node, has been noted. Sudden blue turns in infants with congenital heart disease are often really attacks of paroxysmal tachycardia. Dr. George Sutherland has published cases in which the attacks resemble petit mal.

CARDIAC DILATATION

When gross dilatation is present there is no great difficulty in making a diagnosis, as the apex-beat is noted to be displaced, and there is dullness on percussion well outside the nipple-line. An X-ray will always confirm this. When there is a very slight degree of dilatation only, the diagnosis may be extremely difficult. Slight degrees of rotation of the patient may cause the apex-beat to appear outside the nipple-line; this is a frequent source of fallacy. Percussion is very little help, but X-rays may be of the utmost assistance. The cause of minor degrees of cardiac enlargement may be myocardial infection during a rheumatism or influenza, or it may follow on some severe illness such as pneumonia or typhoid fever. Long-continued anæmia is almost always accompanied by cardiac dilatation. In a certain number of cases showing cardiac dilatation some congenital malformation will be found. The child who is short of breath and who has slight œdema of the extremities, with an enlarged liver and a faint trace of

albumin in the urine, should be suspected of cardiac dilatation. Complete rest, with digitalis and treatment for the underlying condition, is indicated.

CARDIAC HYPERTROPHY

This is best seen following on a damaged pericardium with valvular disease, or in cases of adherent pericardium, or in congenital heart disease. Hypertrophy, as opposed to dilatation, may be suspected from the strength of the cardiac impulse.

CARDIAC MURMURS AND THEIR SIGNIFICANCE

The *cardio-respiratory* murmur is one of the commonest heard in childhood. It is most often found in the thin, nervous type of child, and is best heard on expiration, dying away as the chest is filled, waxing and waning with respiration. It is heard over the whole heart-area, and is probably due to the vessels at the root of the lung being pressed upon by the collapsed lung tissue on expiration, the murmur disappearing as the lung expands and the pressure on these great vessels is removed. There are also other soft systolic murmurs of no significance and unexplained, many being due to slight *congenital malformation*. These are best heard over the basal portion of the heart. *Slight dilatation* of the heart may give rise to transient systolic murmurs which later disappear. This is most commonly accompanied by *anæmia* and may often be termed a *hæmic bruit*. With the disappearance of the anæmia the heart recovers its tone and the murmur disappears.

RHEUMATIC CARDITIS

Inflammation of the valves is most commonly seen in childhood associated with acute rheumatism. The valves damaged are the mitral, aortic, tricuspid and pulmonary valves in that order of frequency. With recent inflammation the murmur is as a rule softer, and as recovery from the damage occurs and the heart's action at the same time is increased the intensity of the murmur is augmented. Loud, high-pitched or musical murmurs suggest a hypertrophy of a heart which is functioning well.

With acute rheumatism a systolic mitral murmur, due to regurgitation at the mitral valve, is most common. As time goes on the valve heals and fibroses, with consequent *mitral stenosis*. A pre-systolic and systolic murmur are present there with a præcordial thrill, and should a fresh attack of the rheumatic infection occur, the heart is inclined to function less perfectly. The murmur, which has been continuously pre-systolic and systolic, with a short, snapping second sound, now changes. The presystolic murmur breaks off and becomes mid-diastolic, so that one hears a systolic, a short, snapping second sound, and then a short mid-diastolic murmur. If compensation becomes even worse and the rheumatism more active with further damage, the murmurs once more change. A systolic murmur is heard and is followed by an early diastolic murmur replacing the second sound. This is the so-called " bellows " murmur. The opening of the bellows represents the systolic and the closure the diastolic murmur. As compensation improves this early diastolic murmur becomes mid-diastolic once more and finally pre-systolic. A child who has had mitral stenosis ought to be in bed as long as there is a mid-diastolic or early diastolic murmur, as this is a definite indication that the infection is active and cardiac compensation poor. For further details regarding treatment *see* p. 446.

Rheumatic pericarditis and adherent pericardium.—This has been dealt with elsewhere, on pp. 453, 454.

Pneumococcal pericarditis.—Pneumococcal or suppurative pericarditis is extremely rare and is seldom diagnosed in life. It sometimes complicates an attack of pneumonia and is discovered at post-mortem, accounting for death in a case which otherwise should have recovered. If diagnosed and drained through the sternum or by removal of a rib with a drainage tube there is a moderate chance of recovery. The author has seen two cases at Great Ormond Street in which this form of treatment has been successful.

CONGENITAL HEART DISEASE (CONGENITAL MORBUS CORDIS)[1]

Congenital heart disease is due to some developmental defect, the cause of which is at present obscure. The diagnosis of the site of the lesion, except in a few clear-cut types, is extremely difficult to make, in fact in the vast majority of cases one can but hazard a guess at the nature of the lesion. The post-mortem examination often shows this to be entirely wrong. The right side of the heart is much more commonly affected than the left side.

Fig. 31.—Clubbing of fingers in congenital heart disease.

1. **Pulmonary stenosis.**—This, next to a patent foramen ovale (p. 230) is the commonest lesion, and gives rise to the most typical clinical picture. At birth, or shortly after, the infant is seen to be blue or cyanosed, with clubbing of the fingers (Fig. 31) and toes and, later, even the nose and lobes of the ears. The respirations are rapid. The lips and tongue are dark-blue in colour and the gums extremely congested. The

[1] G. A. Sutherland, in Garrod, Batten, Thursfield and Paterson's "Diseases of Children," p. 440.

liver is found to be greatly enlarged, even reaching the umbilicus. The præcordial dullness on percussion extends to the right of the sternum, showing the enlargement to be mainly that of the right side of the heart. There is a tendency to epistaxis or sudden hæmorrhage from the mucous membranes. Slight pulmonary infections often prove rapidly fatal.

Pathology.—The pulmonary artery carrying venous blood to the lungs will be found to be stenosed to a greater or less extent; thus there is deficient aeration of the whole of the bloodstream, giving rise to the cyanosis. In an attempt to overcome this the right side of the heart hypertrophies.

2. **Patent inter-ventricular septum.**—This is a much less well-defined condition, there being no particular clinical picture by which it can be recognized. The opening may be small or large; if large there is often no murmur to be heard, but if very small a well-marked murmur is heard over the whole præcordia.

3. **Patent ductus arteriosus.**—In these cases it is always extremely difficult to make a clinical diagnosis. Depending on the degree of patency, there will be many or few symptoms; in fact, many cases of patent ductus arteriosus show no symptoms of ill-health at all. The ductus arteriosus passes from the arch of the aorta to the left branch of the pulmonary artery. Usually it closes shortly after birth, but occasionally it persists. When it does it produces a slight murmur over the whole of the heart and is stated to be present in both systole and diastole (" humming-top " murmur).

4. **Patent foramen ovale.**—This is much the commonest lesion, but it gives rise to little inconvenience. Occasionally there is a soft systolic murmur to be heard during life.

Diagnosis of congenital heart disease.—This may be an extremely difficult matter. As a rule the *murmur* is systolic in time, best heard at the base, and of a much more pronounced and harsh character than those of acquired lesions, except of the severest nature. Often a child, showing practically no disturbance of his

general health, is discovered to have a loud systolic murmur over the heart. An acquired lesion severe enough to produce such a murmur would very seriously disturb the child's general health, and could not possibly have escaped detection. Basal murmurs are much more likely to be congenital than acquired, especially under the age of three.

Cyanosis is present in the majority of severe cases, and is seen most markedly in pulmonary stenosis. Accompanying this there is as a rule a polycythæmia, the red cells being increased to from 6 to 9 million per c.mm.

Clubbing may be seen chiefly in the fingers and toes, but also in the nose and ears, and it is usually present in conjunction with cyanosis in severe cases of congenital morbus cordis. Clubbing is found, however, apart from this condition.

Dyspnœa, or an increased respiratory rate, is present in the more severe cases, and an excellent guide to the severity of the lesion may be obtained by a careful record of the respiratory rate, at rest and after exercise.

Infantilism, or failure to develop, is frequently caused by gross cardiac abnormality. Fifteen per cent. of all mongols have congenital heart defects.

Prognosis.—The infant with a severe congenital heart lesion starts life very much handicapped. Its road is beset with difficulties in the nature of infectious diseases, and it may have to surmount an attack of pulmonary infection. Such infections throw added strain on an already damaged and overworked organ, and a large proportion of infants with the severer lesions succumb in the first few weeks or months of life. Those showing rapid respiration as infants before the crawling or walking stage are unlikely to survive any length of time. Those who appear perfectly healthy but show slight dyspnœa or cyanosis on walking or running may survive for a considerable time, but a careful examination will show that in such cases their compensation is poor, the heart enlarged, the liver congested and increased in size, and the circulation imperfect. The

outlook may be said to be good in those cases where the lesion is discovered by accident in a routine examination by a doctor, or where shortness of breath has been discovered on violent exertion in early or late childhood.

Treatment.—In the infant screaming attacks are common. These may be compared to the *anginal attacks* of the adult. When such an attack occurs the screaming continues for some minutes and then suddenly subsides. This is almost always accompanied by a greater or less degree of constipation. The infant with severe congenital heart disease should never be allowed to become constipated.

Posture.—The position in which the infant is placed to rest in its cot is of importance. Some are comfortable and will sleep well on one side or the other, but there are a certain number who rest only lying across a pillow, with the head hyper-extended.

Gain in weight.—The gain is extremely small in the majority of cases, as owing to weakness and dyspnœa, they are unable to take sufficient at a feed. If breast-fed they tend to underfeed themselves and the breast-milk is prone to fail in quantity. It is well to feed at short intervals, and 7 or even 8 feeds may be necessary in the 24 hours. The giving of concentrated feeds of high caloric value is an obvious advantage. After having failed to gain for some months, or having gained only very slowly, there may be at about a year a marked improvement, many ounces being put on each week. No explanation of this phenomenon can be given. Certainly the more solid the food, that is, the more concentrated, the better the progress made.

Exercise.—Each individual case must be studied before directions as to exercise can be given. As a rule the child will itself tend to restrict its energies owing to its obvious discomfort. The use of a push-chair when out for an airing should be continued in these cases long past the ordinary age.

In the vast majority of cases of congenital heart disease no specific treatment is or will be required throughout life.

CHAPTER IX

BLOOD DISEASES [1, 2]

Anæmia is the failure of the blood-forming tissue to maintain the normal supply of blood-corpuscles and of hæmoglobin to the rest of the body, and its severity is measured by the failure of the capillary blood to reach certain normal standards in respect of numbers of corpuscles and amount of hæmoglobin.

Banti's Disease (Splenic Anæmia)

The cause of this disease is not clear, and it would appear to be a clinical syndrome, chronic enlargement of the spleen with a greater or lesser degree of anæmia. After many years, in adult life, there is a cirrhosis of the liver with jaundice, but probably these two symptoms are not known in childhood. Concurrent with the enlarged spleen, anæmia and cirrhosis, there are repeated hæmatemeses, one of which often proves fatal. In examining the blood, beyond an anæmia nothing is found in the count, the proportion of white cells being normal. For many years it has been denied that this disease occurs before the age of puberty, but we now know that cases do occur at a much earlier age. The author has seen a case at the age of three and another at the age of seven years. The three-year-old case was proved post mortem to have an enlarged spleen and liver, with varices at the lower end of the œsophagus and many outstanding vessels in the wall of the stomach. An erosion of an œsophageal vessel had been the source of the fatal hæmorrhage. The child aged seven had had repeated hæmatemeses with anæmia. The removal

[1] F. J. Poynton, H. Thursfield and Donald Paterson, "The Severe Blood Diseases of Childhood," *Brit. Journ. Child. Dis.*, Vol. 19, pp. 57-80, 128-144 and 178-184.

[2] Garrod, Batten, Thursfield and Paterson's "Diseases of Children," p. 450.

of the much enlarged spleen was followed by an apparent complete recovery.

Acholuric Family Jaundice

This is a congenital disease and is associated with acholuric jaundice. The most characteristic feature is the fragility of the red blood-corpuscles. The envelope which forms the covering seems unduly fragile to any strain.

Etiology and pathology.—The abnormal fragility of the corpuscles is inherited and is transmitted from one generation to another. It occurs in both males and females and is therefore not transmitted through the female to the male as in hæmophilia. The causes of the fragility, the chronic enlargement of the spleen and the recurrent attacks of jaundice, which are not due to bile-pigments, but to blood-pigments, are not known.

Symptoms and diagnosis.—Beyond a certain degree of anæmia, an examination of the blood is negative, and the differential count is normal. The children are sometimes extremely pale, but sometimes the pallor is only slight. In some cases the skin is a little jaundiced, in others markedly so, giving the impression of a mild attack of so-called catarrhal jaundice. The history, however, is that of recurring yellow attacks from the age of a few months, and on examination the spleen is found to be very enlarged, sometimes filling the whole of the left side of the abdomen. The urine is dark, but bile is not present, the colour being due to blood-pigments. An examination of the blood for its fragility, which is diagnostic and as far as we know infallible, shows the corpuscles to be unduly fragile. The "fragility test" is carried out as follows: Some of the blood-corpuscles of the patient and of a control are placed in varying strengths of salt solution. By osmosis weak solutions of salt flow through membranes to strong solutions. The weak salt solutions flow through the envelope of the corpuscle and balloon it up. In the weaker solutions the fragile envelope breaks down, whereas in the stronger

solutions, where the tension is less, hæmolysis does not occur. In the normal blood hæmolysis occurs in a salt

Fig. 32.—Acholuric family jaundice. A. P. and his sister, aged 4 years. The costal margins are marked out and also the spleen.

solution of about 0.47 per cent., whereas in a case of acholuric family jaundice it occurs in solutions as strong

as 0.7 per cent. or more. The corpuscles are otherwise well formed.

Prognosis.—Left to themselves the great majority of these cases live for an ordinary lifetime, but they are miserable and fail to thrive well, they lack energy, are always ailing and unable to lead normal lives. Not all the members of a family are affected, and certainly not in the same degree.

Treatment.—The author is convinced that the proper treatment is to remove the spleen, even in the mild cases. Splenectomy has become such a simple matter and the results are so certain that we are not now warranted in withholding this treatment. In some cases, not in all, the abnormal fragility disappears after operation. Certainly there is no longer anæmia and the patient enjoys normal health.

Typical example of acholuric family jaundice.—A. P., a boy aged 6 years, had been jaundiced and pale from birth, and jaundiced at intervals since. Both the mother and grandmother were said to be jaundiced and pale. There are three children in the family, a younger sister aged 4 years and a brother aged 7 months. The sister has a large firm spleen and her fragility is increased; the brother has a palpable spleen but his fragility is normal. The patient had a large firm spleen, the urine contained urobilin, but not bile-salts or pigment. His fragility was markedly increased. A splenectomy was done, showing a large spleen. The fragility test, although it did not become normal, showed marked improvement after operation, and the child developed a good colour, complete recovery apparently taking place (Fig. 32).

Von Jaksch's Anæmia (Infantile Splenic Anæmia)

This is a type of anæmia of infants accompanied by gross splenic enlargement, with a characteristic blood-count and clinical picture. It is first noticed between the ages of six months and one year.

Etiology and pathology.—The great majority of cases of von Jaksch's anæmia are in premature infants,

twins, or infants who have been fed exclusively on breast-milk for too long. These infants have been short

Fig. 33.—Case of infantile splenic anæmia (von Jaksch's), showing the large spleen and liver.

of iron for a prolonged period, with the result that various organs such as the bone-marrow and the spleen have been making efforts to compensate for this shortage.

This would suggest that infantile splenic anæmia is not a definite disease, but a clinical syndrome, due to a shortage of iron, either congenital or acquired.

Symptoms and diagnosis.—The infant, who is usually premature or one of twins, is seen at the end of nine months to a year because he remains undersized and extremely pale, and shows bossing of the skull, rickety rosary and other signs of well-marked rickets. An examination of the child shows a moderate or gross enlargement of the spleen (Fig. 33). The blood-picture is characteristic, both red cells and hæmoglobin being much decreased, especially the latter. The white cell-count is raised from the normal of 12,000 to 20,000, 30,000, or even 50,000 leucocytes per c.mm. A normal proportion of lymphocytes and polymorphs remains, but many nucleated red cells, both normoblasts and megaloblasts, are present. In fact, this is one of the most characteristic features of the blood-picture. Finally there are from 3 to 5 per cent. of myelocytes present. Except in spleno-medullary leukæmia no other condition occurs in childhood in which myelocytes are present. A higher proportion than 5 per cent. of myelocytes suggests a possibility of leukæmia.

Prognosis.—With careful general attention and feeding the great majority of these children survive. Even in the hospital class of patient 70 per cent. survive.

Treatment.—A well-balanced diet, containing iron in the form of meat juice or underdone scraped steak will be found most useful. Cod-liver oil, sunshine, fresh air, are all essential. The feeding should agree with the age of the child rather than its size and weight, as often the infant diet is too long-continued.

Put shortly, mixed feeding should be persevered with, despite the child's backwardness. For a child one year old 3 grains of the saccharated carbonate of iron should be given three times a day, or 3 drops of Parrish's Food. In severe cases a transfusion is most helpful, 10 c.c. of citrated blood being given per pound body-weight. Recent reports from Ashby of Manchester suggest the efficacy of splenectomy in these cases.

Chlorosis

This is a type of anæmia found at all ages, most commonly in young females at the age of 18 to 20, but also in infants and children who have been kept on a diet lacking in iron and confined without sunshine.

Symptoms and diagnosis.—The blood-count shows a relatively high number of red cells, say 4,000,000, but the hæmoglobin is greatly reduced, say 20 per cent. The white cell-count is normal, with the exception of the presence of nucleated red cells, mainly normoblasts. The common age for the children to be affected is between two and three, but it may be present at any age. The spleen may or may not be palpable, the liver is as a rule enlarged, but rapidly recedes when the child is put to bed and as the anæmia improves. There is a loud hæmic murmur to be heard over the heart, and these children are considered to have congenital or acquired heart disease in many cases. The child is short of breath on exertion, always tired, and in addition to pallor shows a faint lemon tint. The anæmia is most pronounced in the ears.

Treatment.—The giving of iron is specific for this condition. The most successful form is saccharated carbonate of iron, which is given in relatively large quantities, say 10 grains three times daily for a child of from two to three. Parrish's Food, 15 to 20 drops three times daily after meals, is also suitable. Recent work [1] has shown that certain metals having approximately the same atomic weight as iron, e.g. copper and manganese, act as catalysers and cause rapid and more efficient absorption of the iron. When giving Parrish's Food, therefore, it will be found advantageous to give the following mixture :—

Copper sulph., $\frac{1}{200}$ grain
Mang. chlor., $\frac{1}{4}$ grain
Syr. ferri phos. co. ad 1 drachm
Half-a-drachm t. d. s., p. c.

[1] Hart, Steenbock, Waddell and Elvehjem, "Iron in Nutrition : Copper as a Supplement to Iron for Hæmoglobin-building in the Rat," *Journ. Biol. Chem.*, Vol. 77, No. 2, May, 1928.

It is well to put such children to bed, as dilatation of the right side of the heart and congestion of the liver are usually present. Much more rapid progress is made when this rest is insisted on. Sunshine, fresh air and a well-balanced diet (*see* pp. 90-95) are essential.

Acute Lymphatic Leukæmia

This is a disease of the blood and blood-forming organs, the chief signs of which are anæmia and characteristic blood changes. It runs a short fatal course.

Age- and sex-incidence.—It is most uncommon under the age of two and the majority of cases occur between two and five, but cases do occur at any age. It is twice as common in males as in females.

Symptoms and diagnosis.—The child has been noticed to be failing, languid and anæmic, but wasting is not a symptom; in fact most patients look quite well nourished. Pains in the limbs are occasionally complained of, such as those felt in rheumatism. On examination the spleen may or may not be enlarged—quite commonly it is. The majority of cases of the acute type show no glandular enlargement, but in some the glands of the neck, axillæ and groin may be palpable. A hæmic murmur is present, the child is pallid, later becoming waxy and yellow. Petechial hæmorrhages appear on the trunk and limbs and buccal mucous membrane. Occasionally large grey sloughs are seen on the tonsils, which resemble diphtheria. Blood may be present in the urine. A blood examination shows a degree of anæmia. The white cells may be raised or lowered; usually in the earlier stage they are raised to anything from 50,000 to 100,000 and in the later stages it is common to see them drop rapidly down to 1,000. The total of the white cells, however, whether raised or lowered, shows a preponderance of lymphocytes, 95 to 100 per cent. being present. It is this disproportion of the lymphocytes and polymorphs which is the important feature. The lymphocytes are of the small variety and rather immature in their structure. When

the number of white cells is decreased the disease is often called *lymphatic leukanæmia.*

Etiology and pathology.—As to the etiology nothing is known. In fowls a disease similar to this may be transmitted from one bird to another. At post-mortem examinations, beyond pallor and anæmia, nothing characteristic is seen with the naked eye. Hæmorrhages are present in various organs, however. Under the microscope infiltration with lymphocytes can be seen in all the organs, and the kidney, liver, lungs, etc., show masses of lymphocytes throughout.

Prognosis.—No cases recover, although some have exacerbations after which they appear to recover for the time being, but they finally succumb. The course of the disease is as a rule from a few weeks to two months.

Treatment.—There is no treatment for this disease, but arsenic in small doses may be tried. Transfusion undoubtedly prolongs life.

Spleno-medullary Leukæmia (Myelogenous Leukæmia)

This is a type of blood disease characterized by anæmia, enlargement of the spleen and glands, with a characteristic blood-picture.

Age-incidence.—It may occur at any age, two cases having been seen by the author under 4 months old, but the common age is about 10 to 12 years of age.

Symptoms and diagnosis.—Enlargement of the abdomen, with progressive pallor or enlargement of the glands of the neck, are the chief initial symptoms. The spleen becomes extremely large, filling the whole of the left side of the abdomen as a rule. The liver is also enlarged. In infants there is seldom enlargement of glands, but in the more chronic cases in older children the glands of the neck, axillæ and inguinal regions are greatly enlarged. Petechial hæmorrhages are frequently present over the whole of the skin and also in the buccal mucous membrane. Small raised nodules of infiltrated leukæmic tissue sometimes occur over the body and scalp.

Blood-picture.—Although leukæmia may be suspected, the final diagnosis rests on an examination of the blood. The red-cell count shows a moderate degree of anæmia with great lowering of the hæmoglobin. The white-cell count shows an increase of white cells up to several hundred thousand. Of these from 30 to 70 per cent. may be myelocytes. In addition many immature forms of white cells are present and many nucleated red cells.

Etiology.—Nothing is known as to the causation of this type of leukæmia. It is thought by some to be due to an infection.

Pathology.—At autopsy many hæmorrhages may be noted into and over such organs as the kidney, liver and spleen. The mesenteric glands are as a rule greatly enlarged and there may be hæmorrhages into one or more of these. Petechial hæmorrhages are seen over the surface of the lungs. The spleen and liver are both greatly enlarged and a microscopic section shows them to be infiltrated with masses of leucocytes.

Prognosis.—Death invariably results, but in this type, unlike acute lymphatic leukæmia, life may be prolonged for several years.

Treatment.—Arsenic, especially in the form of intravenous injections such as are given in syphilis, is most beneficial and prolongs life considerably. The giving of benzene by the mouth decreases the number of white cells and is also beneficial. Transfusions undoubtedly improve the condition of the child temporarily. X-rays to the spleen and long bones cause a temporary improvement as a rule, but occasionally they bring about a rapid termination. The removal of the spleen, though it has been attempted, has no therapeutic value.

Mixed Leukæmias

Some cases will be found to fall half-way between acute lymphatic leukæmia and the spleno-medullary type; that is, the blood-picture is one showing a large number of myelocytes, but in addition a very high proportion of lymphocytes. The clinical picture

resembles a chronic spleno-medullary rather than the acute lymphatic type of leukæmia. These so-called mixed cases are apparently the link between the two extreme forms.

Lymphadenoma (Hodgkin's Disease)

This is a disease characterized by anæmia, enlargement and infiltration of the spleen, liver and other

Fig. 34.—Lymphadenoma in a boy aged 10, showing massive gland in the neck. (*By courtesy of Dr. Hector Cameron.*)

organs, especially the glands of the abdomen and mediastinum and, sometimes, the neck.

Age-incidence.—It is commonest in childhood between the ages of 10 and 12 years and more common in males than in females.

Symptoms and diagnosis.—Unlike other blood

diseases, Hodgkin's disease causes wasting. The onset is gradual. There are two types: the abdominal type and the type with general glandular enlargement and a high continuous temperature. In the *abdominal type* the child becomes paler and paler, has an enlarged spleen, a certain degree of anæmia and a progressive leucopenia. The liver is also enlarged and there may

Fig. 35.—Lymphadenoma (Hodgkin's disease).

be a few shotty glands in the axillæ or neck. The removal of one of these glands will prove the presence of lymphadenomatous giant cells. The removal of a gland is usually a great help in making a definite diagnosis, but in some cases it is not always possible to make a definite statement from a microscopic section. The size of the spleen is considerable, and it may fill the whole of the left side of the abdomen. It is extremely hard. In the generalized type, where all the glands are enlarged, there may be masses on each side of the

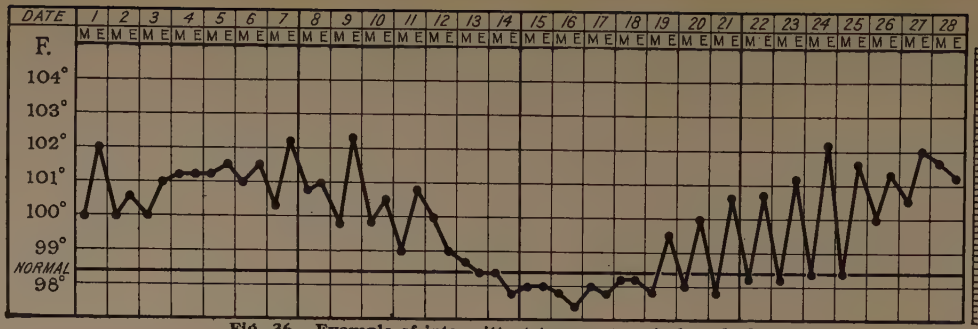

Fig. 36.—Example of intermittent temperature in lymphadenoma.

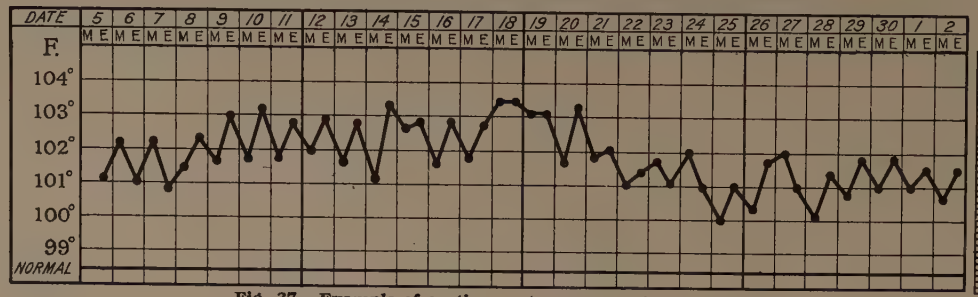

Fig. 37.—Example of continuous temperature in lymphadenoma.

neck (Figs. 34 and 35), soft, almost fluctuating and discrete, reminding one of a bunch of grapes. The removal of one of these glands will usually make the diagnosis simple. If one of the glands removed be cut across and placed in a weak solution of lysol, the solution becomes gelatinous or ropy. As far as the author is aware, this property is shown only in Hodgkin's disease.

Temperature.—In the abdominal type there is as a rule what is known as the Pel-Ebstein temperature. This is an intermittent temperature, up for a week to three weeks and then normal for possibly the same period. During the bouts of fever, the spleen enlarges, the patient becomes much more anæmic, the appetite is poor and all the symptoms are aggravated. If there is a cough due to enlarged mediastinal glands, or if there are pressure symptoms in the abdomen with jaundice and liver enlargement, these also are aggravated. In the generalized form the temperature is either continuously high, 100° to 102°, or it is remittent, up in the evening and down in the morning as a rule.

Differential diagnosis.—1. Tuberculosis must be excluded. Generally the enlarged glands go on to abscess-formation, or the temperature is irregular and not characteristic of lymphadenoma. Removal of a gland easily separates these two diseases.

2. Leukæmia is excluded by the blood-picture.

3. Congenital syphilis is excluded by the Wassermann reaction.

4. In chronic septicæmia, with generalized glandular and splenic enlargement, abscesses are formed.

5. Miliary tuberculosis and typhoid fever are differentiated by the rapid course of the first and by the Widal test in the second.

6. In lymphosarcoma there is as a rule no fever, nor is there much splenic enlargement.

Etiology and pathology.—Nothing is known as to the causation of lymphadenoma. The blood shows moderate anæmia, the white cells are normal or reduced in number, but the proportions are normal. The microscopic pathology shows multinucleated giant cells,

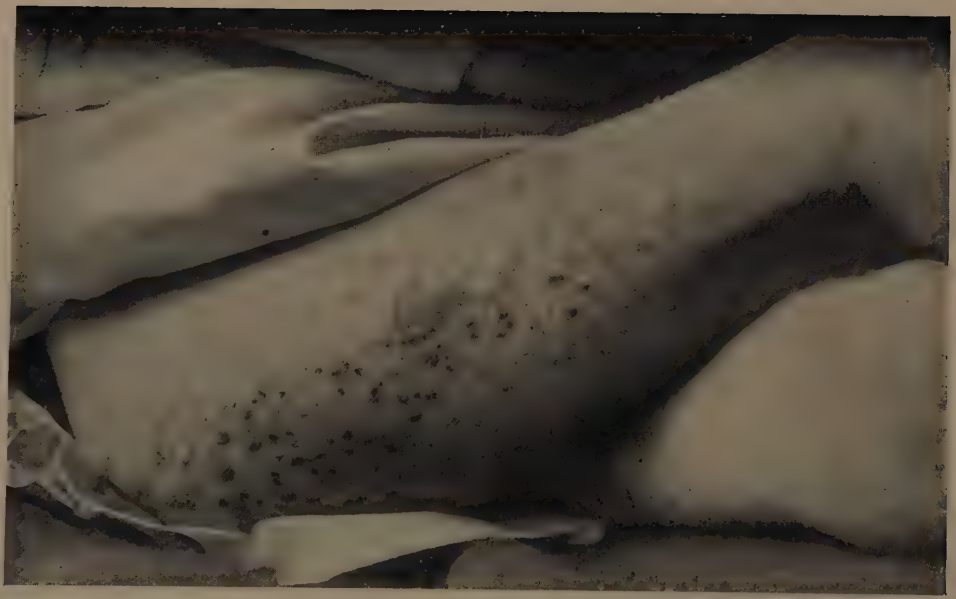

Plate 10.—Rash of purpura.

typical of lymphadenoma. There may be fibrous tissue present in the glands, spleen and liver in some chronic cases. In the liver as well as the spleen there are deposits of epithelioid cells throughout.

Prognosis.—All cases of lymphadenoma end in death, the duration of life being from 1 to 6 years.

Treatment.—X-rays or radium to the spleen and glands are among the most useful therapeutic measures. Arsenic in appropriate doses by the mouth or intravenously has also some therapeutic value, but probably these do nothing more than prolong life slightly. Splenectomy has been performed with no great benefit. It will be found useful occasionally to give transfusions of citrated or whole blood. If the pressure symptoms are great, causing obstruction to the outflow of bile, or pressure on the bowel, or dislocation of the trachea or bronchi, operative treatment may have to be resorted to. All such treatment, however, is merely palliative.

Purpura

As a rule purpura is divided into (1) *purpura hæmorrhagica*, a severe form of bleeding both on the surface of the body and from the mucous surfaces externally; (2) *Henoch's purpura*, where there is bleeding into the wall of the bowel and elsewhere, with sudden attacks of acute abdominal pain, and (3) *simple purpura*, where there are subcutaneous hæmorrhages and possibly hæmaturia. It is clear that there is no dividing line between these groups.

Age-incidence.—Purpura is most common over the age of five, but may occur at any age between a few months and ten years.

Symptoms and diagnosis.—As a rule purpura appears suddenly. It may be over the whole of the body (Plate 10) or largely confined to the mucous surfaces, e.g. the mucous membrane of the mouth, etc. Blood may be found in the motions or in the urine, the latter being of frequent occurrence. In the type described as Henoch's purpura, a common mistake is to consider the case one of acute appendicitis, the abdominal

pain being severe, accompanied by vomiting, a rapid pulse, and tenderness on palpation, but the tongue is clean. As an aid to diagnosis a platelet-count is most useful, as the normal number of platelets, about 300,000 per c.mm., is reduced to say 40,000 or 60,000 per c.mm. Epistaxis may also occur. The urine is often claret-coloured and contains blood-casts as well as blood-corpuscles. In the severe cases pyrexia is present and occasionally the purpura is preceded by vomiting and a rise of temperature. The blood-picture shows a moderate degree of anæmia, with a normal differential white count. The coagulation time is normal.

Prognosis.—In the simple cases the outlook is excellent. Henoch's purpura drags on for some years, but ceases at puberty. In acute purpura hæmorrhagica of the severe type death occurs unless splenectomy is performed (*see* Thrombocytopenic purpura, below).

Etiology and pathology.—The cause is unknown, but it would appear that the sudden decrease in the blood-platelets is an essential factor. This decrease may be brought about by some infection, which in turn causes lysis of the platelets. A microscopic examination of the various organs post mortem shows nothing abnormal beyond a gross degree of anæmia of the bone-marrow. Hæmorrhages may be present on any surface or into any organ.

Treatment.—Since the coagulation time is normal, the giving of horse serum, hæmostatic serum, calcium lactate, etc., to increase the coagulation time of the blood is unnecessary. In severe cases a transfusion of blood is indicated.

Thrombocytopenic Purpura

In recent years Sutherland and Bruce Williamson have separated out a group of cases of purpura characterized by continuous bleeding or tendency to bleed, together with a very low platelet-count. Since life is threatened in such cases, removal of the spleen has been resorted to and has been marked by a dramatic recovery, with a return to normal of the platelet-count. The

work of Sutherland and Williamson has been amply confirmed. This group was probably formerly known as severe purpura hæmorrhagica.

Hæmophilia

Hæmophilia is an inherited tendency to uncontrollable bleeding from wounds or trauma. In the normal individual bleeding would cease spontaneously. The tendency is confined to males.

Etiology.—As a rule a careful family history will show that the males are bleeders and have inherited the tendency from the mother's side of the family. Occasionally an isolated case is found where heredity cannot be conclusively proved. Females are never affected.

Age.—As a rule a child is known to be a bleeder by the age of three.

Clinical picture.—The tendency may appear first at such a minor operation as circumcision or the removal of a tooth, or following an abrasion of the knee. In such circumstances oozing continues for hours or even days, the patient becoming blanched and pale. Occasionally the tendency to bleed may disappear, but as a rule it is present throughout life.

Sites of hæmorrhage.—The hæmorrhage may be due to trauma, when a large hæmatoma will form at any site. The bleeding may occur into the knee- or hip-joint or into the peritoneal cavity. Bleeding from the mucous membrane, however, is commonest, such as from the nose or mouth. Bleeding into the joints is of special importance, as it is difficult to recognize, and may lead to a joint being opened on the assumption that it is filled with pus, tenderness usually being present.

Diagnosis and pathology.—Beyond a moderate degree of anæmia, nothing is found abnormal in the cell-count, and the platelet-count is also normal. The coagulation time is as a rule greatly prolonged. Because of the serious consequences of operating on a bleeder, it is well to interrogate the parents on this point in every case before undertaking operation. In purpura hæmorrhagica there may be bleeding from the gums

and large bruised areas, but in that disease as often as not the patients are girls, the platelet-count is lowered and purpuric patches are as a rule present, this being unusual in hæmophilia.

Prognosis.—The outlook as regards life is not good. Thursfield quotes figures showing that half these patients die before the age of eight, and of those who do survive a number are crippled to a greater or less degree by a partial ankylosis or damage to the joints, secondary to hæmorrhage into them.

Treatment.—The females of bleeder families should not marry if the disease is to be eradicated. Great care should be taken against trauma and abrasions, and no operation should be undertaken without the surgeon being fully aware of the child's condition.

Once the hæmorrhage has started, the best means of stopping it is by pressure, but calcium salts, such as calcium chloride, administered by the mouth or intramuscularly, have some reputation. Horse serum subcutaneously or some of the hæmostatic sera may aid in raising the coagulation time of the blood temporarily. Undoubtedly one of the best modes of treating an acute attack is that of blood transfusion. In such cases as a rule the hæmorrhage ceases. Since the coagulation time is shortened by the application of heat in the normal individual, so it is shortened in cases of hæmophilia, and spraying the mouth or nose with a spray of very warm water or even steam for a moment tends to help coagulation. Hot cloths to a hæmophilic knee-joint are also beneficial. Latterly suggestions have been made that treatment of hæmophilia by protein shock is of value, but the author has no experience of this treatment. Recently Pickering[1] has advocated the giving of quantities of liver to increase the coagulability of the blood and claims marked success in such cases.

[1] J. W. Pickering, "The Recognition, Treatment and Etiology of Hæmophilia," *Lancet*, 1929, i. 1239.

CHAPTER X

DISEASES OF THE GENITO-URINARY SYSTEM[1]

EXAMINATION OF URINE

The examination for defects of the genito-urinary system should always include a routine examination of the urine, and, in suspected cases, the blood-pressure should be taken and hypertrophy of the left side of the heart excluded. Œdema of the limbs and dependent parts of the body should also be looked for. The normal blood-pressure will be found to be from 75 to 85 mm. of mercury in infants and from 90 to 100 mm. up to the age of about 10, after which it is from 100 to 120 mm.

Collection of urine.—There is very little difficulty in collecting a specimen in the male, when, after having washed the penis, a sterile bottle or test-tube may be used. In suspected cases in the female it is much simpler to catheterize, as this is a relatively simple procedure. Failing this, in the tiny infant the wrist of a rubber glove should be fastened with adhesive tape over the vulva, and the urine collected directly into the fingers of the glove. Where a bacteriological examination is required from a female child, as in pyelitis, a catheter specimen should be obtained.

Quantity of urine.—About two-thirds of the fluid intake of the infant is passed per day as urine. At 2 years the output is about 15 oz., at 4 years 20 oz., at 7 years 30 oz., at 12 years 40 oz. The urine is passed very frequently in the young infant, possibly every

[1] J. C. Spence, in Garrod, Batten, Thursfield and Paterson's "Diseases of Children," p. 558.

half-hour or hour. Control is rapidly acquired, however, and by 6 months of age if placed carefully on the chamber after each feed the infant is often dry when picked up to be fed. At one year the child may be able to retain its urine for a period of five or six hours during the night and three hours during the day. At $2\frac{1}{2}$ years the normal and properly trained child should pass its water on waking, after breakfast, at dinner-time, after tea and at bedtime only.

The urine.—A routine examination of the urine should be made in all cases. Not only should it be tested for *albumin* by heating and adding acetic acid, but acetic acid should also be added to cold urine to exclude *globulin*, thus excluding orthostatic albuminuria. For *sugar* Fehling's test should be used. The presence of *acetone* is of special importance, and should be determined by means of Rothera's test, which is performed as follows: To 1 c.c. of urine a crystal or two of sodium nitro-prusside is added and shaken up until it is dissolved. Ammonium sulphate is then added and also a few drops of strong ammonia. If the test is positive the urine then assumes a deep purple colour. *Diacetic acid* is tested for with liq. ferri. perchlor., but the fallacy in this test is that if the patient has been on salicylates the test will be positive. The presence of *pus* or *blood* is best confirmed or excluded by means of the microscope.

A gross fallacy in the testing of the urine may arise from the presence of starch powder such as is commonly used for toilet purposes in the care of infants. Should a little of this powder get into the specimen, a heavy cloud of what appears to be albumin will form on boiling. Albumin can best be confirmed or excluded by filtration or microscopic examination of the urine.

RENAL FUNCTION TESTS

Urea-concentration test.—In the normal individual the urinary output contains about 4 per cent. of urea. Where the kidney is damaged the concentration is reduced, and may be as low as 0.5 per cent. The

technique of the test is as follows : 10 grammes of urea, suitably dissolved and flavoured, are given say at 6 a.m., the urine having been collected immediately before. A specimen is obtained at hourly intervals for say four hours, and the urea content of the various specimens is ascertained by means of Doremus' ureometer, hypobromite solution being used.

Blood-urea test.—Examination of the blood for the blood-urea content is of the greatest value. Normally the blood-urea should fall between 25 and 40 mgm. per cent. In cases where the kidney is damaged the blood-urea may rise to 200 or even 250 mgm. per cent. In such cases some degree of interstitial nephritis is always present.

By some of the newer micro-methods, smaller quantities of blood are required.

Albuminuria

Traces of albumin may be found in the urine of children at any age who are suffering from febrile disturbances, especially diphtheria, whooping-cough and pneumonia. Possibly in such cases there is slight cloudy swelling of the tubules and albuminuria results. Between the ages of 10 and 15 years albuminuria is much more common.

Orthostatic or postural albuminuria

This appears most commonly in children between the ages of 10 and 15 and is slightly more common in boys than girls. It is at this period, when the child is growing rapidly and approaching puberty, that it is most marked. The specimen of urine obtained before rising in the morning is quite free from albumin. As the day goes on each specimen contains a larger and larger quantity, until towards the evening an appreciable quantity is found. On going to rest in the evening the urine once more becomes free. If the child remains in bed throughout the 24 hours, the urine remains quite free, showing that it is due to his being up and about,

or in other words to his posture. In such urine the globulin can be precipitated by the addition of acetic acid, without heating. Serum albumin is also present and is precipitated by heating.

Clinical picture.—The children who suffer from albuminuria are as a rule tall, slim, overgrown and pale, with a tendency to faint easily and get too tired. They stand badly, with the shoulders drooping and the pelvis pushed forward; in fact, the stance is that described under the "nervous-exhaustion child" (p. 352). Headaches are common, especially towards the end of the day, and a variety of symptoms readily attributable to vasomotor instability may be present.

Prognosis and treatment.—Children invariably grow out of this complaint and no particular treatment is necessary for the albumin. Tonics and general toning-up, with plenty of rest and good food, are all that is indicated.

Hæmaturia

This may be due to inflammation of or bleeding from any part of the urogenital tract. *Hæmorrhagic nephritis*, *pyelitis* and *cystitis* are dealt with later in this chapter.

Calculus.—Although extremely rare, calculus does occur occasionally in children. In addition to blood in the urine there is usually some pain. If the calculus is in the bladder the pain is referred either to the bladder or the tip of the penis and is most marked towards the end of the act of micturition. Ureteric calculi are occasionally accompanied by pain, but more often this is absent. In cases of hæmaturia, after a clean specimen of urine has excluded organisms, an X-ray of the urinary tract should be taken.

Renal tuberculosis.—This is very rare in childhood, painless hæmaturia being its chief symptom. The sediment from the urine examined for tubercle bacilli or injected into a guinea-pig will often confirm the diagnosis.

Scurvy.—In this condition blood is invariably

present in the urine, although it may be in such small quantities that it can only be detected microscopically. This rapidly clears up with antiscorbutic treatment.

Blood diseases.—In blood diseases such as purpura and leukæmia, hæmaturia may be a symptom. An examination of the child for purpura elsewhere, and ascertaining the platelet-count and differential count, will readily decide the diagnosis.

Other cases of hæmaturia are due to certain drugs, such as potassium chlorate or hydrogen peroxide.

Paroxysmal Hæmoglobinuria

This is a very rare condition, seen in children usually between the ages of 4 and 10 years. In those cases seen by the author there has always been a positive Wassermann reaction.

Clinical picture.—A child who is apparently quite normal is exposed to a chill, with the result that within the next few hours the urine is quite red with hæmoglobin, the corpuscles having been hæmolysed.

Treatment.—The measures for the treatment of congenital syphilis should be undertaken (*see* Chapter XXI).

Congenital Malformations of the Urinary Tract

These are all of comparatively rare occurrence, and are discovered more often post mortem than during life.

Congenital cystic kidney and *congenital hydronephrosis* are conditions which may be suspected during infancy and which, if unilateral, are amenable to operative treatment.

Ectopia vesicæ is of extremely rare occurrence, and operations for transposition of the ureters into the rectum are in the author's opinion usually unsuccessful. A plastic operation, using the rectus abdominis as a sphincter, is much more likely to be successful.

Hypospadias and *epispadias* present purely surgical problems and are best treated by a plastic operation late in childhood.

Infections of the Urinary Tract

PYELITIS

Since for the diagnosis of pyelitis an examination of the urine is required, this is probably a much commoner disease in infancy and childhood than most practitioners are aware. The difficulties in obtaining a specimen from very young children is sometimes great, and therefore there is probably no infection in infancy in which failure to diagnose occurs so frequently.

Sex-incidence.—Various sets of figures place the proportion of females and males at from 10 to 1 to 5 to 1.

Age-incidence.—Pyelitis may occur at any age from birth onwards. It is infrequent up to the age of 5 or 6 months, when it reaches its maximum and slightly decreases to the age of 5 or 6 years. It is uncommon after this age.

Etiology.—There are three theories of the causation of pyelitis:

1. *That the infection ascends from the vulva.*—The shortness of the urethra in the female and the high incidence of pyelitis in that sex lead to the inference that the vulva becomes contaminated by fæces, and that organisms, B. coli as a rule, pass directly into the bladder and thence to the pelvis of the kidney. This is an attractive and very old theory, which receives very little experimental corroboration.

2. *Direct extension from the bowel to the kidney.*—The incidence of pyelitis in the right kidney is much higher than in the left. Anatomically it is noted that the ascending colon lies directly over the right kidney, and it is thought that a slight abrasion of the mucous membrane of the ascending colon allows organisms to pass by the lymphatics directly into the kidney. In support of such a theory it may be said that prior to an attack of pyelitis there is, as a rule, a history of a few days' digestive upset with loose stools. This cannot, however, be corroborated experimentally.

3. *Infection from the bloodstream.*—In the great majority of cases this is probably the mode of infection.

Plate 11.—Pyelogram of a case of pyonephrosis.

The left kidney is greatly enlarged and both the pelvis and the calyces are much dilated.

It would appear that the infant develops a B. coli septicæmia and that the kidney becomes infected as a result. Some most interesting experimental work on this subject has been done by Miss Lepper.[1] She has shown that if a strain of B. coli which has produced pyelitis in an infant is injected into the ear-vein of a rabbit, and at the same time either the ureter or the renal vein is clamped off, so that the urine is dammed back into the pelvis of the kidney or the kidney becomes congested, then pyelitis occurs. This clamping off need only be very transitory, when the kidney is selected from the whole of the body as the site of infection. It has been known for many years that pyelitis is common where there has been malformation of the urinary tract which dams back the free flow of urine (Plate 11). Kinked ureters, giving rise to hydronephrosis, are present in many cases which subsequently develop pyelitis. This same principle of clamping off the ureter or obstruction to the outflow of urine giving rise to pyelitis is seen during pregnancy, where the infant's head at the brim of the pelvis pressing on the ureters, accompanied by a degree of constipation, causes pyelitis.

It is suggested, then, that the organism gains entrance from the bowel directly into the bloodstream and settles in a congested kidney, the congestion of the kidney being due either to venous stasis or partial hydronephrosis, from obstruction of the urinary tract.

Pathology.—Post-mortem examinations of simple pyelitis are practically unknown, though the pathological appearances are occasionally seen at autopsy by accident. In other words, simple pyelitis does not kill. What one sees at post-mortem examinations is pyelonephritis. In pyelitis the mucous membrane lining the pelvis is slightly pink and suggests a catarrh only, little or no hæmorrhage being seen. In cases of pyelonephritis, however, long rays of pus radiating from the pelvis strike back into the kidney substance. This may be present to a lesser degree in almost every case

[1] Elizabeth Lepper, *Journ. Path. Bact.*, Vol. 23, 1921.

of pyelitis, but in cases which prove fatal the kidney is considerably damaged, both macroscopically and microscopically.

The organism is most commonly B. coli, which accounts for probably 80 per cent. of the cases. Other organisms are staphylococci, streptococci and the typhoid group, occasionally the pneumobacillus.

Symptoms and diagnosis.—The infant appears to have some slight intestinal upset for 24 hours preceding the onset. There may be rather loose, curdled stools or vomiting. The exact moment of onset is in many cases quite clear, being marked very often by a violent rigor. The child turns pale and is seen to tremble, and the mother often suspects a convulsion. The temperature rises rapidly to 104° or 105° and remains above 102°. On examination the abdomen is full, tumid and tender, and the child resents palpation. The clinical picture may vary greatly. The symptoms may be those of some cerebral disease, the child may squint, hold the head back stiffly, or scream continually, so that meningitis is suspected. Other cases appear to be pulmonary, with a high temperature, rapid grunting respiration, but no physical signs in the chest. A last group may simulate abdominal mischief, with a tender abdomen and much vomiting, and appendicitis or peritonitis may be suspected. Yet on careful examination no other disease is definitely indicated and an examination of the urine confirms the diagnosis.

The urine.—From the start an examination of the urine will show it to be full of pus. There may be a certain amount of blood present, but in small quantities only. A trace of albumin is also present. With male infants the child may be fastened up with a test-tube or bottle to obtain a specimen, but with female infants, should pyelitis be seriously suspected, it is always best to catheterize. This is an extremely simple procedure in the female infant, a soft boiled rubber catheter being used. In the tiniest infants a Eustachian catheter will be found most useful. It is necessary to culture the urine always, since the line of treatment adopted

may to some extent be influenced by the organisms present.

Prognosis.—Most cases of acute pyelitis recover, but occasionally one develops pyelonephritis with uræmia, and death follows. The symptoms may subside, but recur again at intervals, the child passing into a state of chronic pyuria. This is commonest in the untreated cases or in cases where there is a gross malformation of the urogenital tract.

Treatment. 1. *Alkalis.*—The usual alkalis are potassium citrate and bicarbonate of soda, and it is well to prescribe them for the infant in equal parts. The dose for an infant of say 6 months is 60 grains of each in the day, to be given as follows :

Pot. cit., 10 gr.
Sod. bicarb., 10 gr.
Syrup, q.s.,
Aq. ad ½ oz.

This should be given four-hourly day and night. The intervals must not be longer than four hours, otherwise the urine will become acid between the doses. By giving part sodium bicarbonate and part potassium citrate vomiting is prevented. When the urine becomes strongly alkaline, which it should in 24–36 hours, the temperature will tend to subside by crisis very like that seen in pneumonia. There is a tendency for a second rise to occur, however, and then a permanent fall. The mixture should be continued for some weeks until a sterile specimen of urine is obtained.

2. *Hexamine.*—This drug is not so efficient as the alkali treatment, except in those cases which are not due to B. coli. Should the temperature remain up, despite the alkali treatment, it will be found as a rule that the organism is some other than B. coli, and hexamine should then be employed. The dose for a 6-months-old infant is 4–5 gr. three times in the day, reduced to 3 gr. three times a day when the temperature begins to fall. Double the quantity of acid sodium phosphate should be given, but not administered at

the same time as the hexamine. They are best given about one hour apart, the acid sodium phosphate being given later. Older children stand hexamine better than younger ones.

3. *Hexyl resorcinol* (*Caprokol*).—This substance is comparatively new and has been used of recent years in the treatment of pyelitis in childhood. It is most efficacious in the chronic form and also for the older children, say from 2 years on. The dose for a child of 2 years is about 1½ gr. three times a day. This dose is dispensed made up in 1 drachm of olive oil, but this disadvantage can be surmounted by emulsifying the olive oil and adding some flavouring agent, as follows :

Hexyl resorcinol, 24 gr.
Olive oil, 6 dr.
Pulv. acaciæ, 2 dr.
Syr. of ginger, 1 oz.
Aq. chlorof. ad 3 oz.

(One drachm of this mixture contains one grain of hexyl resorcinol.)

There is no better urinary antiseptic in chronic pyuria than hexyl resorcinol.

Diet.—The infant should be kept on the diet which it was having formerly, but in slightly smaller quantities and more diluted. The milk may require to be skimmed to prevent accompanying acidosis. In the older child a light fever diet only should be given, during the acute stages ; later, starches such as groats with a very little milk, crisp toast and weak tea for breakfast ; for dinner, gravy and potato and then milky rice pudding ; and at tea thin bread and butter with stewed fruit juice or honey, and weak tea or milk-and-water. Plenty of sugar should be administered. The bowels should be carefully attended to, milk of magnesia for choice being used, either last thing at night or first thing in the morning.

PYELONEPHRITIS

In those cases of pyelitis which go untreated or which are unable to drain properly there is a great

tendency for the infection to pass back into the kidney substance, causing multiple abscesses and giving rise to pyelonephritis. This has all the characteristics of

Fig. 38.–Pyonephrosis, showing dilated pelvis and tortuous ureter.

a mixed glomerular, tubular and interstitial nephritis, and death may take place from uræmic convulsions. Œdema may or may not be present, but in fatal cases it is usually well marked.

PYELOCYSTITIS

Infection of the bladder, along with the pelvis of the kidney, is a common occurrence and is to be expected. Probably in every case of pyelitis there is also accompanying cystitis. During the acute stages it should respond to the treatment given in the cases which are considered to be simple pyelitis.

CYSTITIS

Apart from pyelitis, cystitis is extremely rare. In most cases of pyelitis the bladder also is affected and the condition may be termed a pyelocystitis. In older children the symptoms complained of are frequency and pain on micturition, and the urine is strongly alkaline with an offensive odour.

Treatment.—The bladder should be washed out with a mild antiseptic, such as 1-per-cent. solution of chloramine, or 1-per-cent. solution of Argyrol, or 2-per-cent. solution of Protargol. A weak solution of Condy's fluid is also very useful. Urinary antiseptics by the mouth, such as hexamine, hexyl resorcinol or Cystopurin, are also highly successful.

Enuresis [1]

Before starting the treatment of enuresis it is absolutely essential that organic disease should be ruled out. The urine must be carefully examined to exclude pus, which would suggest cystitis or pyelitis. Again, a trace of albumin in a pale, low-specific-gravity urine suggests interstitial nephritis, with polyuria. If congenital abnormalities elsewhere in the body are present, an examination of the spine for spina bifida occulta should be made. Diabetes insipidus and diabetes mellitus must also be thought of and excluded.

Enuresis of the functional type is the one with which it is proposed to deal.

[1] Donald Paterson, " Hints on the Treatment of Enuresis," *Clin. Journ.*, Aug. 1927.

Symptoms and diagnosis.—This condition is much commoner in boys than in girls and in the great majority of cases a careful history reveals two facts: (1) that the mother or father, usually the latter, wetted the bed as a child beyond the normal period; (2) that the patient is passing his water too frequently during the daytime.

The inherited tendency to poor sphincter-control and hyper-excitability of the bladder-reflex is the most important factor. Contributing factors are:

1. Frequency of micturition during the daytime, so that the bladder holds only small quantities of urine without discomfort.

2. The general health of the child is below par. In considering the child from this standpoint, three possibilities contributing to his ill-health are:

(*a*) Chronic infections, such as bad teeth and enlarged tonsils and adenoids.

(*b*) A chronic state of ketosis or acidosis, produced by overfeeding on fats and relative underfeeding on sugars, or a too liberal diet generally. Very often these children are getting large quantities of milk and eggs, and their sugars have been extremely limited.

(*c*) Physical exertion and mental over-excitement (nervous strain). These children are often the nervous, highly-strung type, who enter into everything in life with great enthusiasm and tend to overtire themselves. They are too much on their legs and too active mentally. Frequently they are "only" children or the youngest in the family.

Treatment and prognosis.—Most of the cases have completely recovered before puberty; in fact after the age of eight very few are met with. As the child gets older, his nervous system tends to become more and more stable and the inherited tendency to poor sphincter-control passes off—he is said to have outgrown it. This tendency cannot be directly affected by treatment, but the contributing factors may be greatly modified by careful management.

1. *The frequency of micturition must be checked.*—This may be accomplished to a great extent voluntarily

by the child itself. If he is very young, emptying the bladder regularly at gradually lengthening periods will tend to distend it slowly, so that it holds larger and larger quantities of urine. The value of this is that when the bladder is properly emptied before going to bed, the child is tided over the night period without requiring to void his urine. To help in this, belladonna is of the greatest service, as it tends to cut off the nervous control of, and therefore to relax, the wall of the bladder, rendering it less irritable. It then fills passively and contains a gradually increasing amount of urine. A suitable dose of belladonna for a child from 2 to 5 years of age would be 5 drops 3 times daily, slowly increased to 10 drops 3 times daily.

2. *Sepsis.*—To improve the general health of the child, sources of sepsis such as carious teeth and enlarged tonsils should be attended to.

3. *The diet* should be gone into and, if too liberal, reduced and the carbohydrates increased in accordance with the Diet Sheet on p. 134.

4. *Rest.*—If the child has not begun to attend school it is well to employ him throughout the morning in some simple kindergarten lessons, so as to keep him off his legs and therefore rest him. Simple games will keep his mind employed without a tendency to wild excitement. Immediately following his midday dinner a rest of an hour should be taken on the bed with blinds drawn. In the afternoon the child should be allowed to romp, and should have a quiet period between tea and bedtime, once more sitting, painting or looking at books. He should go to bed early. If the child is older the régime should certainly include the midday rest and the quiet period after tea, also the early bed-hour. These children tend to overtire themselves to such an extent that on going to bed their sleep is so sound that they fail to respond to their over-distended bladder. In addition their nervous energy is so utterly exhausted that there is insufficient to keep such a muscle as the sphincter of the bladder in constant tone.

5. *Drinks.*—It is well after midday to avoid diuretics such as milk, tea, coffee and cocoa. Plain water or sugar-water ought to be quite sufficient.

6. *Sphagnum moss.*—This has the ability to absorb moisture and the odour of urine. It is especially suitable in cases of enuresis, as it serves the purpose of deodorizing the child's bed and in addition tends to keep the bed dry, leading the child to believe that he is passing less and less urine, and thus he is encouraged. The moss ought to be packed round the child's buttocks and perineum and kept in place by means of light running-shorts, or thin layers should be placed at the level of the buttocks beneath the lower sheet. It can be obtained from W. Martindale, 12, New Cavendish Street, London, W. 1.

Psychological aspect.—One of the most important aspects of this disability is the child's outlook towards his complaint. In most cases, and certainly in the most difficult cases, a too serious view of the question is taken by the mother. The child is cautioned and urged to try to be dry. He is commended when successful and reprimanded when unsuccessful. He is impressed with the importance of overcoming his failing, and with the fact that success or failure in this will make or mar his whole future. All this tends to place a conscious or subconscious strain on the child. An air of optimism about the whole matter must be carefully cultivated by those in charge of the child; indifference should be shown when he fails and praise when he succeeds. He should occupy a room by himself, so that he alone knows when he has succeeded or failed. No discussion by the family or any outsiders should be allowed.

To summarize, the treatment is represented by improving the child's general health, by removing septic foci, seeing to his diet, and providing further physical and mental rest. The psychological aspect of the question must also be kept in mind.

Frequency of Micturition [1]

In infancy, the diet being mainly a fluid one and the quantity large, the bladder must be emptied at frequent intervals. The normal infant passes water after each feed at least, sometimes two or three times between feeds, and several times during the night. As the diet becomes more solid and less fluid, a smaller and smaller quantity of urine is passed. By the age of 18 months control of the bladder by day should be firmly established, and by the age of $2\frac{1}{2}$ years the child should also be dry at night. Frequency of micturition cannot be looked upon as a pathological condition in the young infant, but as one necessitated by the diet.

From birth onward an attempt should be made to establish an association between the act of micturition and the placing of the child on the chamber (*see* p. 15), so that unless he is placed there the child will make a voluntary effort to hold his urine, with consequent enlarging of the bladder and an increased ability to control the sphincter. Lack of training in this simple matter is much the commonest cause of frequency in young children, and this leads very often to enuresis (*see* p. 262). The percentage of bed-wetters and children having frequency is many times higher among the poor than among the well-to-do.

Frequency may be associated with cystitis or stone in the bladder, but both these conditions are rare in childhood. A careful examination of the urine should be made in every case. Strongly acid urine seems to act as an irritant with some children, and the giving of a little bicarbonate of soda or potassium citrate will do away with this. The amount of urine should be carefully measured to exclude diabetes or interstitial nephritis, as in both these conditions frequency is due to the quantity of urine passed.

Occasionally frequency is due to *habit*. The author

[1] Donald Paterson, "Frequency of Micturition in Infancy and Childhood," *Pract.*, June 1926.

has known of cases where children have been in the habit of helping themselves to drinks at intervals throughout the day, with consequent frequency; when taken into hospital this habit has been rapidly broken and the frequency has disappeared.

In cold weather, especially when children are underclad, there is a strong tendency for an increased urinary output, with frequency. In warm weather the exact reverse obtains.

Teething in some children is undoubtedly accompanied by bouts of frequency, probably as a result of the child refusing solid food and taking in much larger quantities of fluid at this time.

Nervous children often show spells of frequency between tea and bedtime, or at any period of the day when they are under nervous tension or much in the limelight. Children going to school for the first time or at a dancing class or when being played or romped with, especially in the presence of strangers, exhibit this tendency. The explanation undoubtedly is the child's fear of an accident. When removed from the exciting surroundings the apprehension at once disappears, and with it the frequency. In all cases of frequency repeated careful examinations of the urine are necessary to exclude the possibility of organic disease.

The greatest possible help is obtained in such cases as are here mentioned by giving a mixture containing 5 drops of tincture of belladonna three times daily, when the wall of the bladder tends to relax and the bladder to hold a larger and larger quantity of urine.

Meatal Ulcer

In male children who are still at the age when they cannot be kept dry both day and night, or in those who are running about shortly after having been circumcised, there is a tendency for an ulcer to form at the external meatus of the urethra. A crust forms and when the urine is passed this is torn off and remains

bleeding, only to re-form before the next micturition. In such cases a great effort should be made to get the child to be dry both day and night. The local treatment consists in the application of a piece of lint to the penis thickly smeared with vaseline, sewn in the pyjamas at night and in the underclothes by day, but kept in place during the whole 24 hours. A little tincture of belladonna is a great help, thereby checking frequency (*see* p. 267).

Pin-point Meatus

Pin-point meatus is of much commoner occurrence than is generally supposed. Although there is very little complaint that the urine cannot be passed, when seen the stream is extremely small and the child is required to exert pressure. In warm weather, with any concentration in the urine of the urinary salts, there is a tendency for smarting to occur at the tip of the penis and irritation or actual pain is experienced, and micturition is dreaded. In such cases undoubtedly a meatotomy is indicated and is a great advantage.

Phimosis

The question as to whether a child requires circumcision is always a debatable one. If there is a long and tight foreskin, so that the glans cannot be uncovered, stretching should be sufficient to rectify this error and allow cleansing to take place. In the author's opinion, however, this is insufficient. Unless the greatest care is exercised, inflammation of the foreskin occurs from its becoming sodden with urine, and this inflammation leads to irritation and a tendency for the attention to be attracted to the penis, with resulting masturbation. Circumcision is better done early than when the child is older, as in the young infant a few days or weeks old extremely little upset is caused, and only a slight degree of anæsthesia is required.

When the foreskin is extremely tight, actual back-pressure and interference with the passage of the urine

may occur, so that dilatation of the bladder and ureters is possible. There is no doubt that the circumcised child can be kept cleaner and is less likely to develop bad habits at an early age than the uncircumcised.

Balanitis

This is usually due to the presence of a long foreskin and lack of cleanliness. The swelling may be great and there is pain on micturition. The treatment is bathing with boracic lotion or applying fomentations, later keeping the part dry with starch and zinc powder. To prevent a recurrence circumcision is advisable.

Hydrocele

There may be a hydrocele of the tunica vaginalis or of the cord. In all cases which persist for more than a few months a hernia accompanying the hydrocele should be suspected. Spontaneous cure is common, and certainly, unless causing great inconvenience, nothing more than simple aspiration should be done until the child has acquired cleanly habits, say at the age of 2 years, when an operation for both hernia and hydrocele is advisable.

Vulvo-vaginitis

The frequency of this condition varies with the climate and the class of child, and is less in cold countries and among the well-to-do. About one-fourth of all cases are gonorrhœal, the remainder being definitely non-gonorrhœal. It occurs most commonly between the ages of 2 and 5 years, but may occur at any age. The seasonal incidence shows it to be most prevalent in summer.

Gonorrhœal and non-gonorrhœal cases.—Among the *non-gonorrhœal* cases the chief source of infection is the fæces, the vulva being infected, especially by B. coli and the intestinal streptococci. Occasionally the diphtheria bacillus and also pneumococci are found. Threadworms, setting up an irritation about the anus and causing scratching of the

anus and vulva, are often responsible for the spread of the organisms.

Gonorrhœal cases are always contracted from some other person, and it is this type of case which tends to spread from child to child in an institution.

Clinical picture.—On examination the vulva is red and swollen, and during the acute stage a thick purulent discharge can be seen covering the labia and appearing over the vaginal orifice. Pressure over the urethra may show a urethral discharge in addition. There is pain on micturition in a proportion of cases. In the non-gonorrhœal type the discharge tends to be less thick and watery and to have an offensive odour. Pain on micturition is also less common in this type. Occasionally pneumococcal peritonitis, which has been described on p. 176, is found complicating the non-gonorrhœal type. In the gonorrhœal type urethritis is a common complication of the vulvo-vaginitis. Gonorrhœal peritonitis or infection of the tubes is rare.

Treatment.—The treatment of non-gonorrhœal cases consists in seeking for threadworms and dealing with these if present, and examining the child for local causes which by giving rise to masturbation may be keeping up infection by trauma. Douches and Sitz baths, using Condy's Fluid in sufficient strength to colour the water pink, are very valuable. Chloramine lotion $\frac{1}{2}$ per cent. is also useful, followed by zinc and starch powder after drying.

In the gonorrhœal group, in addition to Condy's Fluid as a Sitz bath, 2-per-cent. protargol solution may be instilled into the vagina. Acriflavine pessaries, 1-in-500, in coco-butter, are an alternative form of treatment. An excellent method also is to pack the vagina with ribbon gauze soaked in any of the following: 10-per-cent. glycerinum boracis, 10-per-cent. glycerinum icthyol, 5-per-cent. glycerinum eucalypti, one week with each in rotation, or 1-per-cent. picric acid aqueous solution, or 1-per-cent. acriflavine in glycerin or 10-per-cent. protargol in glycerin.

The child should be kept off her legs and given a Sitz bath first thing in the morning, the vagina being packed with half-inch ribbon gauze, which has been dipped in a 10-per-cent. solution of protargol in glycerin, for example. This is repeated once every 24 hours.

A complete cure may take some months, six months being a good average.

NEPHRITIS [1]

Acute Hæmorrhagic Nephritis

The chief characteristic of this disease is the sudden onset of hæmaturia, without reduction in the quantity of urine passed.

Symptoms and diagnosis.—The hæmaturia occurs during or follows some acute or subacute infection, especially of the nasopharynx. At the onset there may be headache, vomiting, abdominal pain and fever, but these may all be attributed to the primary naso-pharyngeal infection. The colour of the urine varies between bright-red and a slightly smoky-brown. The albumin may be accounted for largely by the amount of blood present, both disappearing simultaneously. They may remain for a few days or for some weeks. Blood-casts are present. There is but very slight œdema, if any, and it is largely confined to the face. Neither the blood-pressure nor the blood-urea is raised, and the urea-concentration test shows a normal value.

Etiology and pathology.—Acute streptococcal infections, especially of the nose and throat, are by far the most frequent precursors of hæmorrhagic nephritis. In its commonest form it appears as a sequel to scarlet fever. Pneumococcal and staphylococcal infections are, however, also responsible. Discharging ears, mastoid disease and pneumonia are also sometimes accompanied by hæmorrhagic nephritis. The actual kidney lesion is possibly embolic, secondary to the primary foci referred to, the site of the lesion being in

[1] Donald Paterson and W. G. Wyllie, "Nephritis in Children and its Prognosis," *Arch. Dis. Child.*, Vol. 1, No. 2.

the glomeruli. Once these glomerular vessels have been ruptured there is a tendency for the oozing of blood to continue for some days or even weeks. Statistics show that the condition is equally common in girls and boys, and that it occurs between the ages of 3 and 10 years, mainly between 3 and 5 years.

Prognosis.—More than half the cases recover completely and the remainder retain only a slight trace of albumin in the urine. For practical purposes, therefore, recovery is the rule in this type of nephritis.

Treatment. Rest.—The child should be put to bed and left there until the blood has completely disappeared from the urine.

Septic foci.—Septic tonsils, bad teeth, ear-discharge, mastoid disease, septic spots on the skin and whitlows are all to be carefully searched for and thoroughly dealt with. As far as possible any surgical procedure should be left until the urine has cleared up, but in the case of a discharging ear or a whitlow appropriate treatment should be proceeded with at once. Teeth and tonsils can usually be left until the acute stages of the nephritis are over. There is naturally a fear that if the septic focus is left, the kidney condition may recur, and there can be no doubt that this is what actually happens. Where there are masses of infected glands in the neck, tending to keep up the sepsis and the temperature, great patience must be exercised, and it may be some weeks before it is safe to proceed with the teeth or tonsils.

Diet.—During this acute stage drinks of barley-water, fruit juice and water, sugar-water or Imperial drink should be given in liberal quantities. The child should be offered his meals, consisting largely of starch and milk food, at regular meal-times, that is, at breakfast, dinner and tea-time. For instance, at breakfast cereals with raw or stewed fruit should be given; for dinner vegetable broth and milk pudding; and for tea thin bread and butter or bread-and-milk, or some patent starchy food, such as Benger's Food.

It is difficult to overdo the fruit juice during this

period. A high milk-intake is not desirable and the quantity given should certainly not amount to as much as a pint daily, and that not too creamy.

For the hæmaturia.—With the urine showing only a moderate amount of blood in each specimen with a steady tendency for this to diminish in say a week or ten days, nothing need be done. With large quantities, however, and where there is no tendency towards diminution, the child may become progressively anæmic. Hæmoplastin, 2 to 4 c.c., or horse serum, 20 c.c., given subcutaneously, will be found a valuable hæmostatic in a certain percentage of cases.

For the bowels.—Salines constitute the best treatment, but Petrolagar with phenolphthalein may be given in addition, or cascara or jalap. Metallic purges, such as calomel or grey powder, are best avoided.

Operation.—The removal of the septic focus should not be deferred more than a few weeks after the urine has cleared, or there will almost certainly be a recurrence of the hæmaturia. It is best to proceed with the operation, then, before sending the child to the seaside or attempting to raise his standard of health, as it is during this period that recurrences most often begin.

Mixed Parenchymatous (Tubular) and Glomerular Nephritis

The best-known example of this is post-scarlatinal nephritis, where both the glomeruli and the tubules are damaged. The urine contains not only blood-casts, but also cellular casts from the shedding of the epithelium of the tubules. The symptoms are partly those of purely glomerular nephritis, and largely those described under Acute Parenchymatous Nephritis. The prognosis in such cases lies half-way between the extremely good prognosis of the slight hæmorrhagic nephritis and the grave prognosis of parenchymatous nephritis, or in other words it is moderately good. The treatment is along the lines of that to be described under Parenchymatous Nephritis.

Parenchymatous Nephritis (Nephrosis)

Symptoms and diagnosis.—When the onset is acute the urine is blood-stained and smoky, and albumin is present in large quantities, more than can be accounted for by the amount of blood present. Granular, epithelial and hyaline casts and blood-casts are also numerous. The urine is scanty in quantity. Among the symptoms are headache, vomiting, abdominal pain, slight fever and œdema. Œdema is the most constant symptom of this type of nephritis, and is often generalized.

The onset is usually, however, obscure, with slight generalized œdema, the urine being clear, but containing a varying amount of albumin. At intervals there may be acute exacerbations engrafted on this apparently subacute condition, characterized by smoky urine, much œdema, headache and vomiting.

Œdema.—The face alone may be involved, but much more commonly the whole body is œdematous, the feet, legs, abdomen and dependent parts, particularly the scrotum and the penis or labia, showing œdema in the most marked degree (Fig. 39). The face is often involved to such an extent that the eyes are completely closed.

Urine.—The urine is small in quantity, smoky at first, containing much blood and large quantities of albumin. Granular, epithelial and hyaline casts and blood-casts are present. The urea content is normal.

Blood-urea.—This tends to be raised at first, say to 60 or even 100 mgm. per cent., but to fall to normal, 25 to 40 mgm. per cent., immediately the acute stage is over.

Blood-pressure.—This also is raised at the commencement of the disease, but rapidly falls about the same time as the blood-urea.

Heart.—During the acute phase the heart may be slightly dilated and the beat rapid and forceful.

Etiology and pathology.—A history of a previous sore throat or a rash may suggest that scarlet fever

has been present and has passed unrecognized. This, however, is the exception. In the majority of cases of parenchymatous nephritis the etiology remains obscure, and the most modern theory is that it is due to some toxæmia or biochemical product. During the acute stage the kidney is swollen and engorged, and it is in this stage that the flow of urine is small, probably due to a swelling of the endothelium lining the capillaries of the Malpighian tufts, or to blocking of the tubules by cellular debris. A little later marked cloudy swelling, with fatty degeneration of the tubular cells, occurs. There is a tendency for these damaged cells to be shed, filling the lumen of the tubule, from which they are ejected as an epithelial cast.

Fig. 39.—Acute parenchymatous nephritis. Note œdema of the lower extremities and ascites.

Unless the condition is arrested spontaneously during the acute or early subacute stages it is liable to pass into the subacute and chronic stages, when gross changes appear in the kidney. From the large pale white kidney of the late subacute stage, the small sclerosed fibrotic kidney results.

Prognosis.—During the acute stage the mortality is low, death resulting probably from acute dilatation of the heart rather than from anuria or uræmia. Occasionally, however, anuria is a serious complication and must be treated. The outlook as to complete recovery is poor, probably one-fourth of the cases only showing a complete recovery, the remainder passing into the chronic stage to be discussed later.

Treatment.—1. The patient should be placed between blankets to encourage **sweating.** During the acute stages, that is, while the œdema is severe, he should remain there. Later, when he is allowed to sit up, the greatest care should be taken to avoid chills.

2. **Septic foci.**—A careful search should be made for septic foci. Any bad teeth or septic tonsils should be seen to, the sinuses should be carefully examined and chronic mastoid disease considered. Clausen[1] and Marriott[2] claim that in most cases a septic focus can be found, the removal of which cures or benefits the nephritis. Operation should not be postponed, as there is a likelihood of the disease becoming worse rather than better.

3. **Diet.**—The exclusive use of milk and fresh fruit such as oranges is strongly advocated by certain schools, especially in the acute stages. Certainly a light milk and starch diet is indicated. The restriction of the fluid intake will be found to reduce the œdema, and a salt-free diet also undoubtedly has this effect. Bread made without salt can be obtained from the average baker.

When the disease becomes subacute or chronic (*see* p. 278) a much more substantial diet is necessary, the child requiring red meat, and in fact even more protein than a normal child, if nutrition is to be maintained. Starches in the form of porridge, or Cream of Wheat, for breakfast, vegetables such as potato and peas, and milk pudding for dinner, and bread-and-butter or rusks at tea-time, should be added to the

[1] Clausen, "Studies from the Department of Pediatrics, Washington University School of Medicine and St. Louis Children's Hosp.," 6th series, 1924-25.

[2] Marriott, *Med. Clin. N. Amer.*, 1924, Vol. 7, p. 1413.

diet. It is well to add white meat first, in the form of fish and chicken, then gradually to add red meat as the disease progresses towards the chronic stages. Œdema is not a contra-indication to a high protein or meat diet. Eggs should be given most cautiously.

4. **Medicinal treatment.**—*Sodium bicarbonate and potassium citrate* in doses of from 5 to 10 grains of each, four times daily, will act as a diuretic and will also keep the urine strongly alkaline. That this serves any really useful purpose, however, provided there is no accompanying pyelitis, is most doubtful. Massive doses advocated by Osman,[1] are in the author's opinion a dangerous and unwarranted practice, tending to produce intractable diarrhœa. *Urea* by the mouth in doses of 5 grains twice or three times daily undoubtedly tends to promote diuresis and is well worth attempting. *Diuretin*, 3 grains three times daily, and *pilocarpine nitrate*, $\frac{1}{20}$ grain, have both been tried by the author with temporary and transient benefit only.

Purges.—In order to get rid of the fluid accumulated, drastic purges are necessary and the stools should be watery rather than formed, but not so frequent that the child is thinned. Purges such as *powdered jalap*, 10 to 20 grains, or *liquid extract of cascara sagrada*, a teaspoonful at bedtime, or *powdered rhubarb*, 5 to 10 grains, or *syrup of senna*, a teaspoonful at bedtime, are all suitable, but it is best to avoid *metallic purges*, such as calomel or grey powder.

Tonics.—Iron in the form of *syr. ferri phos. co.* with copper and manganese (*see* p. 239) (Parrish's Chemical Food), half a teaspoonful three times a day, and *ferri carb. sacch.* (sugar of iron), as much as will lie on a sixpence, three times daily, to build up the child, are advisable. Extract of *malt with cod-liver oil* and *iron* should be given during the winter months.

Real or artificial sunlight baths should be given, care being taken to prevent the child from catching cold.

5. **Drainage.**—The introduction of Southey's tubes into the abdominal cavity, legs and abdominal wall,

[1] Osman, *Guy's Hospital Reports*, Vol. 77, p. 386.

will be found most useful. In this way many pints of fluid may be drained away. With such drainage a diuresis is sometimes started and the whole of the fluid from the tissues may quickly disappear. The reason for this and its mode of production are not known, but it has been proved on many occasions. Great care must be taken that no infection is carried in by the introduction of these tubes, as such patients are very prone to develop erysipelas at the site of puncture, or peritonitis when the abdominal cavity is emptied.

6. **Hot-air baths.**—Sweating can often be induced by means of hot-air baths, hot bottles, warm blankets and hot bags. Electric bulbs placed beneath the blankets act efficiently, as also does hot air laid on from a special oven.

It is doubtful whether, apart from tapping, removal of septic foci and dieting, any treatment radically influences the course of this disease.

Subacute and Chronic Parenchymatous Nephritis

Cases of parenchymatous nephritis which fail to clear up in the first few weeks after the onset, pass into a subacute and finally a chronic stage. The urine, which has been scanty, now varies in quantity, and with it the œdema comes and goes. The child remains large and water-logged, and in this state is very liable to contract infections of all kinds, especially pneumonia. Anæmia is almost always present.

Pathology.—Whether the kidney lesion is due to a toxin or to an infection, it would seem to progress one way or the other. In the subacute and chronic cases, the damage extends from the tubules themselves to the surrounding tissue. Symptoms suggestive of the interstitial nephritis type complicate the picture. Thus, as time goes on, the severe œdema disappears; in fact, in childhood very few patients die of nephritis in an œdematous state, except from complications. In time the kidney becomes more and more fibrotic and scarred.

Prognosis and treatment.—The prognosis is usually considered extremely grave, and in consequence

of this various heroic treatments have been attempted, among them Edebohl's operation of decapsulation of the kidney, which in some cases has been successful. Apart from what has been mentioned, the treatment is merely a continuation of that for acute parenchymatous nephritis.

Diet.—Formerly it was the custom to keep subacute and chronic cases of parenchymatous nephritis on a diet mainly of milk and starch. We now realize that a diet high in protein, containing urea and nitrogen, is not deleterious and is possibly beneficial. In this type of nephritis, therefore, the so-called Epstein's diet, which is high in protein, is to be recommended. The meals, then, are similar to those given to healthy children, but smaller in quantity. This policy results in a high intake of urea, which tends to promote a diuresis. In addition it supplies a sufficient quantity of iron and at the same time makes good the loss of albumin which is occurring constantly.

Interstitial Nephritis

For practical purposes this must be considered a chronic nephritis, as we have very little real knowledge of its etiology and onset.

Etiology and pathology.—Interstitial nephritis has been demonstrated accompanying polycystic kidneys. It also follows on untreated pyelitis or pyelonephritis, where the infection has spread back into the kidney substance from the pelvis and on healing has left the kidney substance scarred. Congenital syphilis affecting the kidneys in the first year of life, after treatment may leave permanent damage in the form of a fibrosed kidney. Older children, with repeated attacks of scarlet fever and diphtheria, develop interstitial changes in the kidney. A number of cases, however, have been reported, especially under the heading "renal dwarfism," for which no etiological factor has been ascertained. Microscopically, the tubules are sparse and are being pressed on by fibrous tissue, resulting in a tendency to cyst-formation, the

glomeruli being involved in the scarring. Stained with Van Gieson, the whole kidney shows a great excess of fibrous tissue.

Symptoms and diagnosis.—As a rule the first symptoms are abnormal thirst and the passage of an excessive amount of urine.

Urine.—This is low in specific gravity, 1004 to 1008, exhibits a faint trace of albumin, and appears pale and colourless. The urea-content is extremely low. From 50 to 100 oz. of urine are commonly passed daily by a small child in this condition. The power of the kidney to concentrate urea is poor. Whereas the normal kidney will concentrate from 2 to 4 per cent. in each ounce passed, in this condition 0.5 per cent. is a good average.

Blood-urea.—The normal blood-urea content is from 25–40 mgm. per cent., but in interstitial nephritis it runs from 100 to 200 mgm. per cent.

Blood-pressure.—As a rule the blood-pressure is raised and there is a great tendency to uræmic attacks. The arteries are hardened in many cases, especially at the wrist, and there is hypertrophy of the heart, especially of the left ventricle. Occasionally cerebral hæmorrhage occurs.

Stature.—With any severe degree of interstitial nephritis there is always infantilism. The child is small and as a rule thin and pale-faced. A number of cases of severe late rickets, depending on the kidney lesion, have been observed. It would appear that these cases of so-called renal dwarfism are due to the retention by the kidney of certain salts and the excretion of others, so that the body-salt balance is upset, resulting in rickets. These cases of late rickets show marked bony deformities, such as knock-knee and deformed ribs and arms, just the effects seen in the acute rickets of infancy (Fig. 40).

Prognosis.—In severe cases of interstitial nephritis, giving rise to renal dwarfism and marked infantilism, the patients rarely live beyond 20, the majority dying at or about puberty. Less severe cases, however, without marked polyuria and secondary to repeated

Fig. 40.—A renal dwarf and a normal child, both aged 8.

infections, tend to improve, and life is prolonged much farther. Death is due to attacks of uræmia and cerebral hæmorrhage, or complications, such as pneumonia and infectious diseases.

Treatment. 1. *Diet.*—Undoubtedly protein in all forms should be avoided, as with the introduction of more urea-producing foods into the diet there is a gradually increasing tendency for uræmia to occur. Red meat and eggs should be avoided, and even fish and chicken should be given in small quantities only. The diet consists mainly of cereals and milk. For breakfast the child should have porridge, groats, Cream of Wheat, Force, Puffed Rice, etc., with raw or stewed fruit juice and milk; for dinner, gravy or broth, with plenty of vegetables, and rice, tapioca or semolina pudding or stewed fruit; and for tea-supper, macaroni or spaghetti or some other cooked cereal, or bread-and-butter with jam or honey and milk.

2. *Drinks.*—The child should have plenty of extra drinks between meals. Limiting the fluid seems to aggravate the condition; in fact extra drinks may have to be given during the night.

3. *The bowels* should be kept well open by the measures mentioned under parenchymatous nephritis (p. 274).

4. *Artificial sunlight* and *cod-liver oil* are both indicated.

5. *Minor uræmic attacks*, appearing as bilious attacks, should be carefully watched for, the eyes and the heart examined periodically, and the blood-pressure tested at intervals.

6. As a *tonic* Parrish's Chemical Food (*see* p. 239) or syr. ferri iodidi, half a teaspoonful three times a day, is indicated, or extract of malt and iron.

7. The *Wassermann reaction* should always be resorted to in order to exclude congenital syphilis, which, if present, must be cautiously treated with arsenic. Syphilitic nephritis is described on p. 482.

CHAPTER XI

DISEASES OF THE NERVOUS SYSTEM

EPIDEMIC ENCEPHALITIS (ENCEPHALITIS LETHARGICA)

This condition, popularly known as "sleepy sickness," may be defined as an acute inflammation of certain portions of the brain and brain stem, giving rise as a rule to squint, stupor and various sequelæ. Although the disease appears to be a new one in the British Isles, references to it have been made for many years. The first series of cases among children in England were described by Still and Batten[1] in 1918.

Etiology and pathology. Age-incidence.—Children may be affected at any age, almost from birth onward, but the disease cannot be said to be common in childhood. Of 25 cases analysed by the author[2] the youngest was 3 months and the oldest 11 years old, but the majority were more than 3 years of age.

Sex-incidence.—The disease is equally common in males and females.

Seasonal incidence.—Unlike poliomyelitis, it is much more prevalent during the winter months.

The cause of epidemic encephalitis is not clearly understood, but some observers, by employing a virus obtained from human cases, have produced similar brain lesions by inoculation in rabbits and monkeys. The infecting agent appears to be a filter-passing virus. The chief portions of the central nervous system affected

[1] F. E. Batten and G. F. Still, "Epidemic Stupor," *Lancet*, 1918, i. 686.

[2] Donald Paterson and J. C. Spence, "The After-Effects of Epidemic Encephalitis in Children," *Lancet*, 1921, ii. 491.

are the basal ganglia and the nuclei of the third nerves, but any part of the brain may be damaged. Microscopically such lesions show a perivascular infiltration with lymphocytes, and, in the acute stages, a greater or less degree of hæmorrhage and congestion of the affected area.

Clinical picture.—The length of the incubation period of this disease is not known, but its onset is

Fig. 41. Case of amentia following on encephalitis lethargica.

usually accompanied by fever, drowsiness, headache, double vision and often vomiting. The child may lie in a stuperose or drowsy condition for a few days or even for weeks. He can be roused, but soon relapses into his drowsy state. The speech is slurring, and in the cases seen in 1921 catatonia was characteristic. When asked to put out the tongue or hold up a hand,

the child would continue in that position for many minutes if left alone. Ophthalmoplegia or squint is one of the most characteristic symptoms.

The clinical picture varies greatly. The drowsiness may resemble that of tuberculous meningitis, or there may be no drowsiness at all, only extreme irritability. Occasionally the pain in the abdominal muscles or the

Fig. 42.—Post-encephalitis lethargica. Note the obvious mental defect depicted.

back is the chief symptom at the onset, and this has given rise to the mistaken diagnosis of appendicitis or peritonitis. Examination of the cerebro-spinal fluid shows it to be under pressure, and a slight increase in the albumin is usually found, with an increase in the cells varying from 10 or 15 to as many as 100 per c.mm. The sugar is, as a rule, increased in amount, and the chlorides are normal.

After a period varying from a few days to a few weeks the temperature settles and the child appears to have made a recovery.

Course of the disease and prognosis.—Very often up to this period no diagnosis has been made, but as the child begins to recover he may show a great change in character. Diurnal somnolence and nocturnal wakefulness may begin, the child being wide awake, noisy, active and perhaps dirty in his habits during the night, but sleeping soundly during the day-time.[1]

Mental changes.—The majority of these children show some signs of mental change, and often they are morally incorrigible.[2] Those who were truthful and pleasant become the reverse; lying and thieving habits are also observed. In some cases it is merely that a bright child has become dull and backward at school, but this, especially in infants, may reach such a degree that the child becomes an idiot.

Late sequelæ.—Reviewed after the lapse of some years, the disease will appear to have progressed after the acute stage had been passed. Motor paralysis and Parkinsonism appear. Myoclonus may be present either in the acute stage or may appear as a late sequela. Respiratory symptoms, where the child has "puffing attacks," may develop. After a spell of violent breathing there is apnœa, followed by another "puffing attack." Sometimes during these attacks the child may appear to faint or may actually have a convulsion. Lesions in some portions of the brain result in a picture indistinguishable from that of Fröhlich's syndrome (the fat child). Epidemic hiccough, which has been shown to be a manifestation of this disease, is extremely uncommon in children.

Differential diagnosis.—During the acute stages the chief difficulty is to distinguish this condition from meningitis (*see* Table XIV, p. 295). Lumbar

[1] H. Morley Fletcher and J. D. Rolleston, *Brit. Journ. Child. Dis.*, Vol. 18, p. 69.

[2] Cyril Burt, "The Young Delinquent" (University of London Press, 1927).

puncture is of the greatest assistance. The clinical picture much more resembles that of tuberculous meningitis than one of the acute pyogenic forms, but because of the rapid onset tuberculous meningitis can usually be ruled out. The squint may suggest a brain tumour or a peripheral neuritis such as post-diphtheritic neuritis. An examination of the eyes as a rule shows them to be normal, but a few cases have been reported showing optic neuritis. Fever does not usually accompany a brain tumour, and palatal paralysis accompanies the squint and loss of accommodation when it is due to diphtheria.

Ultimate prognosis.—The outlook as to life is good, those who are grossly backward mentally and damaged physically succumbing to intercurrent infections. In only a small proportion of cases, varying from 25 to 30 per cent., does there seem to be complete recovery. In 18 out of the 25 cases studied by the author there was gross or moderate mental defect. In those studied by Dr. Mary Stevenson[1] only 3 out of 65 had recovered completely. All the sequelæ, apart from Parkinsonism and mental deterioration, would appear to improve, but these two show a progressive course.

Treatment.—**During the acute stage,** rest in bed is essential. Lumbar punctures are always beneficial, not only for diagnostic purposes, but also for purposes of treatment. Great *nursing care* is required, as there may be incontinence, and during the stuperose stage the child may develop bed-sores. A water-bed will be found a very great help in the nursing. Should swallowing be difficult, the child must be fed nasally or by means of a gastric tube. The mouth and eyes require special cleansing.

Drugs.—In the restless cases chloral combined with a little bromide may be given at intervals; paraldehyde in doses of 30 minims rectally is also most efficacious. Hexamine should be administered, 5 grains five times daily, as an antiseptic for the cerebro-spinal fluid.

[1] Mary M. Stevenson, *Arch. Dis. Child.*, Vol. 3, No. 14, p. 57.

Salicylates, in the form either of aspirin or of sodium salicylate, are useful as an antipyretic.

Diet.—The usual fever diet should be given, together with copious fluids. The times of administration of the starchy foods will depend on the readiness or otherwise of the child to take them. Owing to difficulty in swallowing or to refusal of the food from other causes, frequent small quantities may have to be given.

In the chronic stages each type of sequela may require a different form of management, the severe cases often requiring institutional treatment. Here, undoubtedly, educational measures should be regarded as the basis of treatment. Special nurses for backward children, tutors and specially instructed governesses may all play a useful part in re-educating the child and teaching self-control and discipline. There is little doubt that there remains great scope for advance in the treatment of the after-effects of encephalitis lethargica, which presents some of the most trying, difficult and depressing cases encountered in the whole range of pædiatrics.

Other Forms of Encephalitis

Post-measles and post-vaccinal encephalitis are known to occur in childhood; they are considered at pp. 395 and 415.

MENINGITIS

Tuberculous Meningitis

This disease may be briefly defined as a miliary tuberculous infection of the brain and cerebral meninges.

Age-incidence.—The youngest case the author has seen was of a child aged 3 months. The incidence rises rapidly and reaches its maximum between one and two years, that is during the second year of life, then falls rapidly to the age of seven, when it is a comparatively uncommon disease.

Etiology and pathology.—For practical purposes tuberculous meningitis is always secondary to some old primary focus. As a rule the initial lesion is in the bronchial lymph-glands or mesenteric glands. In only one of 70 autopsies[1] was the primary focus not easily recognizable; in 87 per cent. the bronchial glands were caseous, but 58 per cent. had caseous mesenteric glands in addition. The bacillus of tuberculosis may be inhaled or ingested, and it is probable that most cases of tuberculous meningitis are infected in one or other of these ways by the human bacillus. Bovine tuberculosis is more often glandular, secondary to infected tonsils or infections of the bones, and the mortality from bovine tubercle is low in comparison. The milk supply is usually the source of infection in such cases.

Example.—Two girls, aged 4 and 6, were admitted to the Hospital for Sick Children, Great Ormond Street, and died within three weeks of each other. Neither had had a previous illness of any sort, but for 12 months previously to this a lodger with active pulmonary tuberculosis had been living in contact with them.

The *seasonal incidence* of miliary tuberculosis shows it to be most common in the first quarter of the year. Sunshine is almost absent, confinement is great, vitamins are difficult to obtain, infections are at their worst; possibly these are the factors which cause the primary focus to spread and become miliary.

The post-mortem appearances show that the brunt of the infection has fallen on the choroid plexus and basal meninges. The meninges over the vertex may show nothing more than œdema and have a rather gelatinous appearance. An examination of the meninges and vessels running into the Sylvian fissure, however, shows them to be invariably studded with fine pearl-like tubercles, and the whole of the choroid plexus is a mass of similar tubercles. Very often, in examining such

[1] Donald Paterson, "Tuberculous Meningitis—Is it a Preventable Disease?" *Pract.*, June 1923.

a brain, small tuberculomata may be found embedded in its substance (Plate 12), and the question arises whether these may not have been the primary source from which the infection has spread directly to the cerebro-spinal fluid in many, if not all, cases. A varying degree of internal hydrocephalus is usual.

Symptoms and diagnosis.—The clinical picture is not always clear-cut, as miliary tuberculosis in some other part of the body may overshadow the meningeal infection. Thus if the brunt of the infection has fallen on the lungs, the case is taken to be a pulmonary one, while at the same time the meningeal infection would pass unnoticed. The meningeal symptoms are ushered in by lassitude, headache, drowsiness and irritability. The child who was formerly active wants to be nursed or carried about, and may be noticed to drop off to sleep while playing with his toys, a thing quite unusual for him. *Constipation* is usually present and all the early symptoms are generally attributed by the parents to this fact. An *occasional vomit* may occur, not necessarily in relation to food, but vomiting is not usually a prominent symptom. *Hyperæsthesia* is prominent during the early stages and manifests itself in a complaint of pains and tenderness wherever the child is palpated. In answer to leading questions, abdominal trouble or mastoid disease may be suspected, but a more careful examination will show this apparent tenderness to be a general hyperæsthesia. As the *irritable stage* passes into the *stage of compression* the pulse becomes slower, the headache more intense and a high cephalic cry may be given at intervals. During the second week the child shows signs of rapid wasting, the abdomen is scaphoid, he lies curled up on his side, dislikes a light and may pull the bedclothes over his head. There may be slight stiffness of the neck muscles but no definite head retraction. The reflexes may be exaggerated or diminished and Kernig's sign is of little value, for it is often absent. The child may appear to frown or knit the brows, looking thoughtful, probably because of the headache. If he is roused he answers a

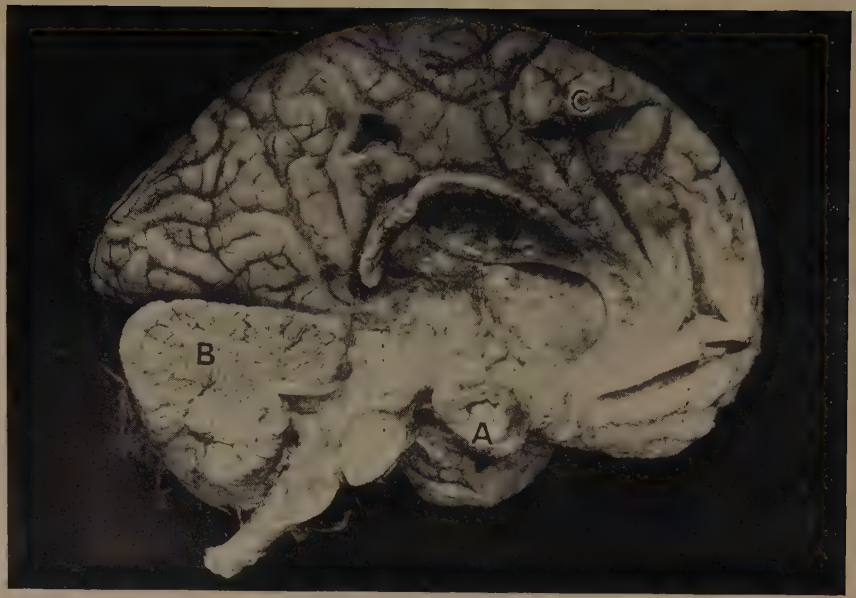

Plate 12.—Brain of a child ten months old, showing tuberculous masses at A, B, and C.

question sensibly, but almost before he has finished speaking drops off to sleep again. The third stage is that of *coma*, and shows a tendency for the temperature to rise and the pulse to quicken. Tache cérébrale is more definite, flushing and sweating occur and fine tremors of the hands are noticeable. The pupils become dilated and may be unequal, the breathing is intermittent, the limbs are spastic and the child may have a series of convulsions. A squint is common and the eyes are wide open and staring. Swallowing ceases, the temperature rises to 106° or 108°, and death follows. The length of the illness depends on the age and nutrition of the child, the average time being about 21 days, but one will do well to be guarded about its length. Too much stress should not be laid on any one symptom in the diagnosis—the general picture rather should be considered.

Differential diagnosis.—A child who is merely constipated may simulate the early stages closely, and the child with a bilious or acidosis attack may vomit and be constipated, but as a rule the vomiting is too severe for that of tuberculous meningitis. Differential diagnosis may be impossible, however, without lumbar puncture. Poliomyelitis, polio-encephalitis, encephalitis lethargica must all be thought of carefully, and a lumbar puncture will help in diagnosis. A local examination will exclude chronic mastoiditis, and cerebral abscess should be thought of. Brain tumours have a slower and more insidious onset even than tuberculous meningitis.

Cerebro-spinal fluid.—This is clear, and under pressure a cobweb-like clot forms on standing, and the fluid contains from 15 to 200 cells per c.mm. The cells are lymphocytes. The sugar is reduced and the albumin is increased up to 0.1 per cent. from the normal 0.025 per cent. The chlorides are reduced to as low as 0.6 grm. per cent. from the normal 0.725 grm. per cent.

Prognosis.—In all cases of definite tuberculous meningitis the outlook is hopeless.

Treatment.—Repeated lumbar punctures may be done where this relieves the headache. Chloral and bromide should be given in full doses, and hexamine may be given by the mouth in doses of from 15–30 grains per day. Careful nursing will prevent the mouth and eyes from becoming sore. For the convulsions the administration of a little chloroform will give great relief.

Prevention.—Children who are below par, showing lassitude, wasting, lack of appetite, occasional rise of temperature for no apparent reason, dry skin and hypotonia of the muscles, should be treated as potential tuberculous patients. They should be given a holiday with fresh air, sunshine and good food, and should be so built up that the primary lesion which may have developed may tend to be completely healed or shut off. When one sees how rapidly the incidence of this disease falls after the age of 3 years has been passed and how comparatively rare it is after the age of 10 years, one sees at what age the greatest care should be concentrated to prevent the primary lesion becoming miliary.

Cerebro-spinal Meningitis (Meningococcal or Postbasic Meningitis; Spotted Fever)

Age-incidence.—This disease, though much commoner during the first year of life, may also be met with in later childhood.

Etiology and pathology.—Cerebro-spinal meningitis is due to infection with the meningococcus, a Gram-negative diplococcus which is, as a rule, intracellular. The starting-point of the infection and its mode of entrance is not known, but from recent work it would appear that the infection commences in the choroid plexus and ependyma, and the ventricles are first filled with pus, this draining into the subarachnoid space and infecting the cerebral and spinal meninges. At autopsy the ventricles are found full of pus, but in addition much pus is found over the brain surface,

especially the basal portion, which is matted, and the basal foramina tend to be occluded. The commonest sequel to a mild postbasic meningitis is an internal hydrocephalus, due to such a blockage occurring. The more severe forms cause acute inflammation of the meninges with an increased intracranial pressure and actual inflammation of the brain substance itself, proving rapidly fatal or leaving much gross permanent damage. From the pathology as described it would appear that in the early stages there is a meningococcal septicæmia.

Clinical picture.—The disease commences suddenly with a high fever, vomiting and, in little infants, looseness of the stools, a feature which may for a time distract attention from the cerebral condition. In the older child intense headache is complained of. It is in the infant that the basal portion of the brain is the part mainly affected and that the clinical picture of postbasic meningitis is most commonly found.

Fulminating type.—In these cases, usually occurring in older children and severe in form, death may occur during the septicæmic stage. Purpura occurs with extreme toxæmia and massive cerebral infection.

Mild (Type 3) infection in older children.—The temperature is high, the headache intense, the neck is found to be stiff, with definite head-retraction, occasionally to an extreme degree, and as a rule Kernig's sign is present. The disease differs from tuberculous meningitis in that there is no photophobia. The child is conscious and able to talk, presenting quite a different picture from the acute symptoms observed in pneumococcal and streptococcal meningitis. Vomiting occurs, but not frequently. The abdomen tends to be scaphoid. In some older children the symptoms are curiously mild, amounting to nothing more than a slight rise of temperature and headache. The temperature then settles for several days, rising again at intervals. In one such case this continued for some months and ultimately the child recovered completely.

Postbasic type.—In infants this is the form commonly taken. Following on the loose stools there is a rise of temperature and marked head-retraction (Fig. 43). This may be so extreme that the head actually approximates to the sacral region. The fontanelle bulges, and within a few days the basal region becomes so matted that drainage ceases and internal hydrocephalus results.

Diagnosis.—The diagnosis cannot be definitely made except by examination of the cerebro-spinal fluid. The history, however, is of some help. The saying " three

Fig. 43.—Postbasic meningitis. Note the marked head-retraction. (*By courtesy of Dr. Hector Cameron.*)

days for septic meningitis, three weeks for tuberculous meningitis, three months for cerebro-spinal meningitis" is essentially true. This, in so far as it refers to cerebro-spinal meningitis, however, merely means that as a rule the disease is not so severe that it kills quickly. An examination of Table XIV will show the differential diagnosis from other forms of meningitis and from poliomyelitis and encephalitis.

Treatment and prognosis.—In the *fulminating type*, where a meningococcal septicæmia is being dealt with and massive purpura is present, there is very little hope of recovery, as these cases are so badly infected that nothing seems of any avail. In the *milder type* it

TABLE XIV

ANALYSIS OF SYMPTOMS OF MENINGITIS, POLIOMYELTIS AND ENCEPHALITIS LETHARGICA AS AN AID TO DIAGNOSIS

	Head retraction	*Photophobia*	*Bowel movements*	*Temperature*	*Cerebro-spinal fluid*				*Length of life*
					Albumin	Sugar	Cells	Chlorides	
Septic meningitis (staphylococcal, streptococcal, pneumococcal).	Well marked.	Not present.	Nothing characteristic.	High.	Increase to 0.1 or 0.2% at once.	Absent.	Many polymorphs present.	Normal.	3 days.
Cerebro-spinal meningitis (meningococcal, postbasic).	Very marked.	Not present. Eyes wide staring.	Inclined to looseness.	High.	Moderate increase say to 0.1%	Decreased or absent.	Many polymorphs.	Normal.	Say 3 months or recovery.
Tuberculous meningitis.	Slight stiffness only.	Well marked.	Definite constipation.	No temperature or very slight.	Slight increase, say to 0.05 or 0.075%	Decreased or absent.	Slight increase, say 25 to 100 lymphocytes.	Decrease from normal 7.5 mg. % to 6.0mg.%	3 weeks.
Poliomyelitis.	Definite neck stiffness.	Not present.	Nothing characteristic.	High at first.	Very slight increase during acute stage.	Unchanged.	Very slight increase of lymphocytes.	Normal.	Recover as a rule.
Encephalitis lethargica.	Slight occasionally.	Not a feature.	Nothing characteristic.	High at first.	Not increased.	Present and often increased.	Slight increase, esp. lymphocytes.	Normal.	Recover as rule.

would appear that complete recovery can occur if the disease is treated at an early stage. Lumbar puncture should be done during the acute stage twice daily. The fluid should be allowed to run off until it ceases to flow from the needle, and should then be carefully measured. If possible the type of meningococcus should be ascertained, Type 3 being the mild one. The appropriate serum should be injected intrathecally where this is possible, employing about 5 c.c. less than the quantity of cerebro-spinal fluid withdrawn. As the temperature falls and the fluid becomes clear and the headache less severe, one lumbar puncture only need be done daily, until finally an occasional puncture only is necessary. Lumbar puncture, however, should be continued as long as the organism can be cultured from the fluid withdrawn and its cell-content is raised. Where the case is diagnosed late and the pus is thick and fails to run from the needle, cistern punctures should be attempted (*see* p. 320). In such cases the prognosis as to complete recovery is not so good.

Postbasic type.—The infant with postbasic meningitis presents a serious problem. Th mortality is extremely high, because of the tendency for the bulk of the infection to be basal and for communication between the interior and exterior of the brain to be cut off. The foramina of Magendie and Lushka are obliterated early and internal hydrocephalus rapidly develops. If the diagnosis can be made shortly after the onset and lumbar puncture persevered with, the results generally are good. Intrathecal administration of the appropriate serum is called for.

Intravenous serum therapy.—Since the point of infection is the choroid plexus and ependyma, there is no other means of reaching the site of infection than by the intravenous route. Giving serum in this manner to infants, however, is difficult, especially since it must be given on several occasions. Direct injection of the serum into the ventricle may therefore be resorted to, or a vessel at the ankle or bend of the elbow may be utilized. For intravenous injection 10 c.c. of the

appropriate type or of a polyvalent serum should be diluted in normal saline. Subsequent doses must be given well within seven days to prevent shock. As a rule a considerable degree of collapse occurs after this form of therapy and there is much upset to the patient in administering it.

Subcutaneous and intramuscular serum therapy.—In addition to the administration of serum by the bloodstream and intrathecally, it may be given beneath the skin or into the muscles. It is best to give 20 c.c. of the polyvalent serum subcutaneously. This may be repeated in two or three days and possibly a third time. After extensive serum therapy there is always the possibility of severe serum sickness, with painful spots and a serum erythema.

Drugs.—The best-known antiseptic is hexamine, 5 gr. three times daily for a child of 2 years, and a smaller dose for infants.

There is no disease in which wasting occurs so rapidly as in cerebro-spinal meningitis. Careful nursing and feeding are therefore essential. Food should be given to the limit of the child's powers of digestion. A careful watch should be kept on the tongue and bowels for signs of intolerance. During the febrile stage cream and fats should be avoided, and a diet similar to that described on p. 134 is suggested for older children.

Streptococcal or Pneumococcal Meningitis (Septic Meningitis)

This may be either primary, or secondary to some other focus in the body; it is caused by a pyogenic organism, such as the streptococcus, pneumococcus or staphylococcus.

Etiology and pathology.—Whether septic meningitis is ever primary is a matter of debate. Certainly it is most commonly secondary to some very definite focus, the source of which is most often discovered at post-mortem. By far the most common primary source is the ear. An infected middle ear involves the mastoid

bones and may spread by actual erosion of the bones to the dura itself. It is from this infected dura that the meningitis is rapidly contracted. There may be for a time a localized meningitis, but ultimately the meninges become infected throughout. A post-mortem examination in such a case shows a thick felted layer of pus, especially over the vertex of the brain, but most marked in the region of the ear if this organ has been the primary focus. It will be noted that the pus has infected the spinal meninges as well. Sections of the brain show it to be congested, and a certain amount of actual cerebral inflammation or encephalitis is always present. Occasionally septic meningitis is the result of a primary focus in some other part of the body, such as tonsillitis, infections of the sinuses, peritonitis, empyema, pneumonia, pericarditis or arthritis, the organism being either the pneumococcus or the streptococcus. Accompanying these infections there is often a septicæmia, the organisms being transported to the meninges in this manner. Cerebral abscess, a rare sequel to both middle-ear disease and bronchiectasis, ultimately causes death, as a rule by a spread of the infection to the meninges.

Clinical picture and prognosis.—In a typical case a child whose ear has discharged for some time becomes feverish and irritable and vomits ; within a few hours he is delirious and very definite head-retraction can be made out. The presence or absence of knee-jerks or plantar reflexes in a young child is not of great help, but delirium, unconsciousness and head-retraction are most significant when the onset is sudden. The temperature continues at 103° or 104° throughout the illness. The disease takes an extremely rapid course, lasting as a rule not more than three days. By the second day small tremors and convulsions, with twitchings of the eyes, may be noted. These convulsions later become constant and general.

Speaking generally, cases of streptococcal, pneumococcal or staphylococcal meningitis are fatal. There have been a few authentic cases of recovery, but in

such cases the infection has been mild and the cerebro-spinal fluid never typical.

Diagnosis.—Apart from the clinical picture, the diagnosis is best made by examining the fluid after lumbar or cistern puncture. The fluid is seen to be quite opaque and milky and, examined under the microscope, it is crowded with pus-cells which themselves contain chains of streptococci or pneumococci. The sugar has disappeared and the fluid is grossly pathological; later on in the disease it is so thick that it cannot flow from the needle.

Treatment.—Lumbar puncture is indicated, and if an early diagnosis is made the source of the infection, whether in the ear or elsewhere, should be investigated and dealt with, if necessary by operation. Hexamine should be given by the mouth, and an effort should be made to sustain the child's strength by feeding, employing a stomach-tube if necessary. Some authorities suggest washing the subdural space with saline, an inlet and exit having been made through the skull. It is questionable, however, whether this is a practical method of treatment.

Influenzal Meningitis

Etiology and pathology.—This is a rare disease which affects children at any age, but mostly those aged between six months and one year.

The organism involved is Pfeiffer's bacillus, and in the early stages of this disease it can be cultured from the blood. The meninges and surface of the brain are grossly involved, a thick layer of felted yellow pus extending over the whole brain surface, especially that of the vertex. The spinal portion of the meninges is also affected, but to a much less extent. The influenza bacillus would appear to be pleomorphic, tending on culture to form Gram-negative coccal forms which may resemble the meningoccocus to some slight extent. This possibility of error has been pointed out many times in the literature.

Clinical picture.—There is an acute onset with a high fever, vomiting, rigor and a tendency to squint, or to convulsions. In every respect it resembles the onset of septic meningitis (*see* p. 298). Head-retraction is seldom a very pronounced symptom. The fontanelle bulges.

Prognosis.—Influenzal meningitis appears to be almost uniformly fatal, and certainly in infants is always so. The disease lasts about 10 days and is marked by convulsions. The cerebro-spinal fluid becomes thicker and thicker, until finally nothing can be obtained by either lumbar or cistern puncture. The fever continues high and swallowing is increasingly difficult.

Treatment.—Beyond lumbar puncture and careful nursing and feeding there is no rational treatment.

Drugs.—Hexamine should be given, 1 grain four-hourly being a suitable dose for an infant.

ACUTE ANTERIOR POLIOMYELITIS (INFANTILE PARALYSIS; HEINE-MEDIN DISEASE)

This is an acute infection of the anterior-horn cells of the spinal cord, giving rise to motor paralysis.

Age-incidence.—The disease most commonly affects children between the ages of one and three years; after the age of three the incidence rapidly falls.

Seasonal incidence.—In the British Isles the disease is most common during the months of July, August, September and October, especially August and September. Sporadic cases do occur, however, at intervals throughout the year and may at times assume the proportions of a minor epidemic.

Etiology.—This disease has been shown to be produced by a filter-passing virus. It can be produced and transferred in monkeys by intrathecal, intraperitoneal, intravenous or subcutaneous inoculation with the virus.

Incubation period.—Experimentally the incubation

period varies from 8 to 12 days. Clinically it would appear to be from 2 to 14 days, as a rule less than 7 days.

Mode of spread of infection.—Most commonly it is spread by carriers who are themselves quite healthy. No fresh cases have occurred from contact with cases admitted to the Great Ormond Street wards for many years. It is doubtful, therefore, whether the disease is ever contracted from contact with a patient. It may possibly be transmitted through the milk supply.[1]

Incidence of cases.—It is an extreme rarity to find more than one case in any one family, but there are a number of instances on record of 2, 3 or more cases occurring in one household. Large numbers of cases may occur in institutions and schools.

Pathology.—The essential lesion is a destruction of the anterior-horn cells of the spinal cord. There are two factors, the first hæmorrhagic, the second toxic. The onset in some cases is extremely sudden, probably due to extensive hæmorrhage in and around the anterior-horn cells. In other cases, where the onset is more gradual, there is a minimum of hæmorrhage and a maximum of actual cell destruction. During the acute stage the naked-eye details of the surface of the brain may show nothing beyond slight congestion. On section of both the brain and cord at the site of the lesion, however, marked congestion is easily visible. This is particularly apparent in sections of the cord in the cervical and lumbar enlargement, both being especially liable to damage. In the later stages the hæmorrhage having been absorbed, areas of sclerosis are visible. The affected site of the cord is usually much diminished in size. If the brain itself is affected a crenated or walnut-like appearance is seen. Occasionally cysts are present. Microscopically, in the acute stages the whole cord is seen to be hyperæmic and congested. In the anterior-horn region the vessels have actually burst and there is extravasation of blood into

[1] Discussion on Epidemic Poliomyelitis, *Lancet*, Dec. 3, 1927, p. 1190.

this region. Red blood-corpuscles and lymphocytes are scattered about the anterior-horn cells. A little later the anterior-horn cells are found to stain badly, showing commencing destruction and finally only indistinct remnants can be seen. It would appear that the lymphocytes present act as "scavengers," absorbing their effete remnants. The destruction of these anterior-horn cells may be only partial or it may be complete at various levels. The later picture microscopically shows sclerosis and contraction in the anterior-horn region.

Muscles.—With disuse rapid fatty degeneration and atrophy of the muscle-fibres takes place.

Clinical picture.—The onset of this disease is always sudden. Cases are recorded in which paralysis occurred in a few moments. Commonly the child goes to bed at night apparently all right, and in the morning he is found to be paralysed. Most often, however, there is an onset with fever, headache, possibly a vomit, sometimes pain all over, diarrhœa occasionally, and rigidity of the neck. After 48 hours to three days the temperature settles and it is discovered that there is paralysis of some muscle or muscles. In two out of every three cases there is fever, and in almost every case pains in the limbs exist for some days, even for as long as a month. As a rule there is rigidity of the neck, and almost invariably during the earlier period the child objects to being sat up, that is, to having the thighs flexed on the abdomen.

Classification of the various types.—Clearly the clinical picture will show great variations according to the site of the lesion. In cases where the lesion is high up in the spinal cord and involves the brain itself, there is much more likelihood of meningeal symptoms than in cases where the lumbar enlargement alone is involved. It should be noted that when the motor cells of the cord are destroyed, groups of muscles rather than individual muscles are picked out.

The *spinal form* of paralysis is by far the most frequent and most typical. In this type the destruction

of the innervation to the leg muscles is most common, paralysis of the anterior tibial group being the most common of all. However, one or both legs, one or both arms, the diaphragm, the intercostals, the muscles supporting the spine either in front or behind, the shoulder-girdle or the neck muscles may all be involved, and any combination of these is possible. Once the acute stage is over the muscles become flaccid. On taking up the affected limb and letting it fall it has a dead feeling, and on grasping the muscles between the finger and thumb they are softer than the normal. Voluntary movement is impaired. If the pons or medulla is damaged, some of the cranial nerves may show paralysis, the sixth and seventh most frequently, especially the seventh. If the cervical region of the cord is damaged the arms or the shoulder-girdle or the neck muscles may show paralysis, with the result that the head is held up with difficulty. Where the thoracic segments are involved, paralysis of the abdominal and intercostal muscles occurs, in which event there may be a great bulging of the abdomen on the flaccid side. Where the muscles supporting the spine are involved great curvature may follow. Very occasionally almost all the muscles, except those necessary for life, such as the respiratory muscles, are involved and permanently damaged.

Batten has described a *neuritic form*. Whether such a form exists separately or not is difficult to say. Accompanying most cases of poliomyelitis, certainly all of the spinal form, there is definite pain on movement of the affected part and a dislike on the part of the patient to flex the thighs on the abdomen. The child also dislikes sitting up for some days. The congestion of the whole of the cord, however, is so extreme, and the influence of the toxins so widespread, that there can be little wonder if the sensory nerves and roots are involved to some slight extent in most cases. The pain may last up to 30 days after the onset.

In the *abortive type*, especially noticeable and characteristic during epidemics, a child who has been in

contact with or in the same district as other cases will develop a temperature with headache, vomiting, stiffness of the neck, injection of the throat and pains in the limbs. After a period of a day or two, however, all these symptoms subside and, as far as can be ascertained, complete recovery takes place. It is suggested that these are abortive cases of poliomyelitis. Had there not been an epidemic, they would have been diagnosed as influenza.

Batten describes in addition an *ascending and descending form*, where the paralysis, having involved one limb first, extends to all parts of the body, often proving fatal. He also describes a *bulbar and pontine form*, where cranial nerves are picked out, especially the seventh, involving the facial muscles. The *cerebral and cerebellar forms* really represent what may be termed *polio-encephalitis*. Occasionally they give rise to hemiplegia. If the frontal region is affected the mentality is impaired ; if the motor region is involved hemiplegia may result. If the occipital cortex is damaged sight may be lost. In the cerebellar forms slight or gross ataxia may result. Finally Batten describes a *meningitic form*, which in its signs and symptoms closely resembles tuberculous meningitis. The clinical picture is that of a severe polio-encephalitis or high spinal lesion.

Diagnosis.—Mistakes in the diagnosis are not commonly made, but an error may occur in differentiating acute rheumatism from poliomyelitis. In acute rheumatism, however, all movements can be made, there is often an accompanying heart lesion, and the affected joints may be red or swollen. Scurvy also may cause confusion, but blood in the urine and swelling of the gums are differential points of significance. An examination of the cerebro-spinal fluid during the acute stage will practically always determine the diagnosis. The fluid shows normal chlorides, normal sugar, and a slight increase both of albumin and of lymphocytic cells.

Prognosis.—The immediate prognosis depends on the site of the lesion. If the respiratory centre is

involved or the nerve supply to some vital muscle, such as the intercostal or diaphragmatic muscles or the muscles of swallowing, then death will follow rapidly. The average death-rate in an epidemic is about 10 per cent. It is said that about half the cases during an epidemic are of the abortive type and recover completely. The definite diagnosis of abortive cases will always be difficult. Of cases which are definitely recognized as poliomyelitis only a small proportion recover completely. Batten says that " of those cases which are left crippled, some have weakness of one or both legs and arms, with hemiplegia, with mental defect, squint, loss of sight or permanent ataxia." To aid in prognosis it may be said that cases in which the onset of the paralysis is sudden often recover more completely than those in which the paralysis takes some time to declare itself. This is probably due to there being a great degree of hæmorrhage and a lesser degree of permanent toxic damage in the former group. The prognosis as to the length of the limb is extremely difficult to form. Sometimes when there is but very slight muscular damage it has been noticed that the limb ceases to develop or grow in length, whereas even with very extreme muscular damage the bones of the limb may develop normally. The greatest amount of improvement occurs within the first six months, after which it becomes extremely slow or ceases.

Treatment.—During the acute stage the patient should be kept absolutely at rest. Any movement of the limbs or of the child itself must of necessity pull on the inflamed spinal cord or cause it to move, with the possibility of further hæmorrhage. In order to prevent deformity such as foot- or wrist- drop, the affected limbs should be supported in the most comfortable position, and at the earliest moment that the pain will permit, suitable splints should be applied. If the lower limbs are involved it is sometimes desirable to use a cage, keeping the weight of the bedclothes off the toes.

Serum.—In Australia, New Zealand, and parts of

America the intrathecal injection of serum obtained from a convalescent patient is advocated and practised. Whether it has a beneficial effect must remain a difficult point to decide, as by the time the diagnosis has been made the giving of the serum cannot possibly prevent damage ; if given at an earlier stage it cannot be known whether paralysis might have otherwise occurred. The obtaining of such a serum and its administration are both difficult, but where possible this method of treatment should be employed. The giving of the serum subcutaneously is also of great value, and should be employed where intrathecal injection is impracticable.

Antiseptics.—Hexamine has the reputation of appearing in the cerebro-spinal fluid as an antiseptic. In small doses it is harmless to any patient. In larger quantities it causes cystitis in children and therefore is contra-indicated. It may be given in doses of 2 to 5 grains three times daily to a child of from 2 to 5 years.

Subacute and chronic stages.—Once the pain has passed off from the limbs, which have been previously splinted, massage should be started. This is done in order to prevent gross wasting of the muscles by disuse, so that meanwhile the motor cells may have an opportunity to recover. Passive movement with massage and electrical treatment are beneficial in this stage, that is from the third or fourth week onwards. Later, light non-inflammable celluloid splints, made from plaster casts, should be obtained, the affected part in this way being kept in perfect position. The splint should be removed for active and passive movement at intervals. The care of an experienced orthopædic surgeon is a great help in adjusting the splints and thereby preventing deformities later. In the chronic stage suitable tenotomies may need to be done from time to time to undo contractures. Recovery or improvement may take place up to a period of one year. As circulation in the affected part is impaired and the limb is cold and blue, great care should be taken to maintain warmth, and the parts may require to be warmly clad both in winter and summer.

Polio-encephalitis.

Etiology.—Polio-encephalitis is a virus infection identical with that of poliomyelitis, with the same incubation period and age-incidence.

Pathology.—The site of the lesion is the chief point of difference from poliomyelitis, the brunt of the damage falling on the brain, especially the basal nuclei and the pons and medulla. A common sequel is either hemiplegia due to gross hæmorrhage along with the infection, or bulbar paralysis.

Clinical picture.—Commencing with a series of convulsions, such cases may later develop hemiplegia. In a recent group of cases seen by the author bulbar symptoms predominated, with slight light-headedness, but chiefly there was inability to swallow or to speak plainly and a marked tendency for the saliva to drip from the mouth as a result. A post-mortem in one such case showed gross hæmorrhage lying over the surface of the pons and medulla. Another and similar case partially recovered, leaving difficulty in speech. Several other cases recovered completely. In such cases the same symptoms of malaise and feverishness as are found in poliomyelitis are present.

Treatment.—This should be along similar lines to that of poliomyelitis (*see* p. 305).

PROGRESSIVE SPINAL MUSCULAR ATROPHY (WERDNIG-HOFFMANN PARALYSIS) [1, 2]

This, a very rare condition, is familial and independent of sex. It occurs chiefly in infants, the first symptoms being usually noticed shortly after birth, though in some cases they do not appear for a few months.

Clinical picture.—A healthy infant is noticed to be unable to move its limbs properly in the proximal parts, that is at the shoulders and hips, while the hands

[1] J. G. Greenfield and Ruby O. Stern, "The Anatomical Identity of the Werdnig-Hoffmann and the Oppenheim Forms of Infantile Muscular Atrophy," *Brain*, Vol. 50, p. 652.

[2] Donald Paterson, *Westminster Hospital Reports*, Vol. 20, 1929, p. 43.

and ankles, elbows and wrists move freely and the grip is good. Later it is noticed that the diaphragm tends to overact, the abdomen being large and pendulous (Fig. 44), whereas the intercostals are acting feebly. The grip in the hands and the movement becomes less and less good, until the child is only able to move the toes slightly. The facial muscles and power to swallow are unimpaired. The neck muscles are weak, so that

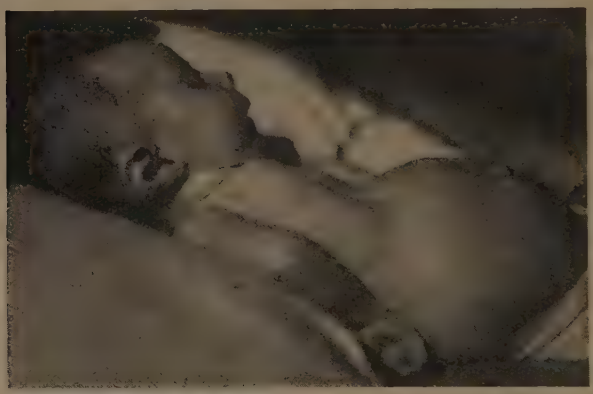

Fig. 44.—Werdnig-Hoffmann paralysis (progressive spinal muscular atrophy). Note the over-action of the diaphragm causing protrusion of the abdomen with sucking in of the sternum. The arms hang in a lifeless position, due to their almost complete paralysis.

the child is unable to hold up its head. Death takes place after some weeks or months from some complication such as pneumonia. The duration of the disease and age of onset vary greatly, some children living to the age of 2 or 3 years, the process being extremely chronic.

Etiology and pathology.—The cause is unknown. At post-mortem nothing is seen with the naked eye, but microscopically the anterior-horn cells show slight or gross destruction. They are few in number and are small and show absence of the dendrons. Roughly speaking the picture resembles that of a chronic anterior

poliomyelitis, if such a condition exist. The muscles are everywhere deficient or absent, the pectorals and glutei being mere thin sheets.

Treatment.—There is no treatment.

Amyotonia Congenita

This rare condition has a pathology similar to that of progressive spinal muscular atrophy (Werdnig-Hoffmann paralysis) just described. The characteristic feature is that the child is born hypotonic, flabby, weak and unable to raise the head or make any movements, and that there is a steady progress towards recovery, so that ultimately in a proportion of cases the child is able to get about and do moderately well. The treatment required consists of massage, electrical treatment, sunbaths and general tonics and good feeding.

It would appear that despite the wide divergence between the clinical picture of amyotonia congenita and Werdnig-Hoffmann paralysis, in view of the similarity of the pathology these two diseases must be one and the same.

PERIPHERAL NEURITIS

Erythrœdema Polyneuritis (Pink Disease)[1]

This condition, which was first described by Swift in Australia, is characterized by symptoms of a peripheral sensory neuritis.

Clinical picture and mode of onset. Age.—It may be seen between the ages of 4 months and 3½ years, but is most common between 9 months and 2 years. There is a history of an *indefinite feverish cold* with slight bronchitis. This seems to improve, and after a *quiescent period* of two to three weeks the secondary symptoms commence. These are as a rule termed "teething," the child being *nervy, restless and irritable.* The picture

[1] Donald Paterson and J. G. Greenfield, "Erythrœdema Polyneuritis (The so-called Pink Disease)," *Quart. Journ. Med.*, Vol. 17, No. 65, Oct. 1923.

of *abject misery* he presents is most characteristic (Fig. 45, 1). There is *failure to sleep* and *refusal of food*, so that the parents become worn out.

Rash and neuritis.—This is characteristically seen on the *hands and feet*, extending as far as the wrist, and also on the *nose, forehead, cheeks and chin*. It may be so marked that the term "raw-beef hands" is applicable. The condition of the hands and feet waxes and wanes. There is a fine desquamation on the palms and soles. There is *marked irritation over the whole body* and the feet feel *cold, clammy and damp*. The *extremities are very irritable* and in the older children there is a complaint that they *burn and itch*. To gain relief the child is continually rubbing his feet or hands one against the other or against the bed-clothes, and his *attitude in bed* is extremely characteristic—hunched up or crouching on the hands and knees, the face being buried in the pillow and constantly rolled from side to side (Fig. 45, 2). *Photophobia* accompanies this stage of the disease and the eyes may be tightly shut. When the child is turned on its back its mouth hangs open, like that of a young gosling, due to a pronounced *hypotonia of all the muscles of the body*. *Small ulcers* may be seen in the cheeks and on the tongue and occasionally the *teeth become loose* and appear in the stools. The *hair falls out*, leaving bald patches and *nails may loosen* and drop off. The *knee-jerks disappear* at some stage of the illness. *Anæsthesia* over the extremities is marked and a needle can be pressed into the feet and hands without notice being taken of it.

Smell.—Some of the cases have a mouse-like odour about them.

Temperature.—As a rule the condition is afebrile when fully developed, but in the early stages there is a slight fever.

Tachycardia.—One of the most characteristic features is the rapidity of the heart-beat throughout the illness, without any satisfactory explanation. This alone is so characteristic that it should be an important point in the diagnosis of the disease (*see* p. 225).

Etiology.—The condition does not appear to be seasonal, but is apparently confined to certain districts. As many of the cases are breast-fed, this

Fig. 45.—Pink disease.

1. Look of extreme misery on child's face.
2. Attitude of the child in bed.

is strong evidence against its being a deficiency disease. Diphtheria plays no part in it, but there is possibly a connexion with influenza.

Pathology.—The examination of the nervous system shows a peripheral neuritis and chronic inflammatory changes in the spinal cord and nerve roots, in which the sensory nerve fibres are affected more than the motor.

Prognosis.—Recovery is complete in from 3 to 9 months. Death may, however, take place from intercurrent infections and starvation.

Treatment.—This consists largely of treating the symptoms. It is most difficult to cope with the anorexia. The clothing should be light to prevent sweating, and gloves and socks should be worn to prevent scratching with the hands and feet. Small doses of arsenic and strychnine may be given at short intervals. Artificial sunshine and changes to the seaside are to be recommended. The diet should be well-balanced and include vitamins.

Lately Findlay and Stern have shown experimentally that a disease very like this may be produced in young rats and cured by the administration of raw potato or white of egg.[1] The author has not had an opportunity of trying this treatment.

Undoubtedly the most important factor in the treatment of a case of pink disease is good nursing.

Post-Diphtheritic Paralysis

This is a form of peripheral neuritis, manifesting itself as a rule by palatal paralysis and loss of knee-jerks and of eye accommodation.

Etiology.—Post-diphtheritic paralysis may occur at any age, but is most commonly seen in children between 4 and 8 years old. It is a sequela either of a very severe diphtheritic infection, or of a mild one which has been untreated or treated very late in the course of the disease.

Clinical picture.—Some four to six weeks after the attack, which usually has been entirely missed, the child is seen to be unsteady on his legs. Next it is

[1] G. M. Findlay and Ruby O. Stern, "A Syndrome in the Rat resembling Pink Disease in Man," *Arch. Dis. Child.*, Vol. 4, No. 19, p. 1.

noticed that fluids tend to regurgitate through his nose, and finally that the voice is nasal in character. A clinical examination reveals :

1. *A local lesion* at the site of the infection. If the infection is nasal or pharyngeal, the palate is involved ; if it is an infection of the vulva, penis or anus, the perineal muscles are involved.

2. *A specific lesion.* In all cases of diphtheritic neuritis eye accommodation is affected, it may be to a great degree or only slightly. For instance, if the child is reading a book, it is noticed that he puts it down to rest at short intervals ; this is because his power of accommodation is impaired.

3. *A general lesion.* This is a general affection of the nerves throughout the body. The knee-jerks are lost, the diaphragm or the intercostals may be affected, interfering seriously with respiration and the power to cough.

The duration of the illness is from two to six weeks.

Complications.—When the diaphragm or intercostals are seriously affected the child is most prone to respiratory infections and to massive collapse of the lungs. Coupled with this there is a difficulty in swallowing due to palatal paralysis, and there is a great tendency to aspiration-pneumonia on this account. One of the most unpleasant complications is vomiting, and it may be necessary to feed rectally for the time being, with a solution containing 7 to 10 per cent. of glucose. Feeding with a nasal tube or stomach tube, small quantities at a time, is often useful for a short period.

Sudden death from suprarenal hæmorrhage is recorded in a certain proportion of these cases.

Both in the acute stage of diphtheria and also as one of the sequelæ, cardiac failure may occur. Sudden death is apt to take place in a small proportion of cases from this cause.

Prognosis.—Gross post-diphtheritic paralysis, especially when the diaphragm or intercostals are involved, is of serious import, claiming a high mortality.

Treatment. Antitoxins.—As a rule, when the diagnosis of post-diphtheritic paralysis is made, some weeks have elapsed, and in some cases antitoxin has already been given. In cases where it has not been given, a swab should be taken of the nose and throat, and in addition from 8,000 to 20,000 units of antitoxin may be administered; no very great benefit should be expected from this, as the damage has already been done to the peripheral nerve.

General treatment.—The child must be nursed as flat as possible, thus throwing little work on the heart, but when the diaphragm or intercostals become involved and swallowing is difficult, the head should be placed in such a way that the mucus does not tend to collect in the back of the throat. Constant swabbing with gauze on forceps helps in this respect.

Diet.—This should be fluid or semi-solid, depending on the ease or difficulty with which the child is able to swallow. Very often semi-solids are managed best, milk thickened with some starchy preparation such as groats or Benger's Food being most successful. Feeding by means of a nasal tube in difficult cases is most useful, and can be carried out comparatively easily.

Drug treatment.—As a nerve stimulant strychnine is most efficacious and may be given hypodermically at four-hourly intervals during the acute stage. The dose is from $\frac{1}{400}$ gr. to $\frac{1}{100}$ gr. for children between the ages of 2 and 8 years. In the earlier stages for children of this age it may be exhibited best in the form of tincture of nux vomica, 1 to 3 minims, three times daily.

Atropine is useful in such cases, to dry up undue secretion and, in addition, to cut off the cardiac depressors. The dose for children between the ages of 2 and 8 years is from $\frac{1}{400}$ gr. to $\frac{1}{100}$ gr.

Cardiac stimulants such as digitalis are not indicated, but brandy may be administered during the acute stages.

The essential in these cases is careful expert nursing, which is more important than the actual treatment.

As the palatal paralysis clears up and the power of accommodation improves, the knee-jerks returning, the child should be allowed to have an extra pillow and finally to sit up and get about. Massage to the limbs at this stage is valuable. It may take 6 or 8 weeks or even longer before the paralysis has improved enough to get the child up. A careful watch should be kept for signs of heart-block and cardiac irregularity. In the later stages general tonics, sun-baths and fresh air are strongly indicated.

Facial Paralysis

Occasionally this condition is congenital. As a rule when present at birth it is due to pressure by forceps. Later, the most common cause is middle-ear disease, such as otitis media. Sometimes it is said to be due to " catching cold " in the nerve, for instance through sitting in a draughty carriage with the cold air blowing on one side of the face. Again, it may be due to injury during a mastoid operation. Septic teeth may give rise to infection of the nerve somewhere in its length. Poliomyelitis or polio-encephalitis affecting the facial nerve is not common, but it does occur, the author having seen several cases. Cerebral tumour and meningitis should be thought of as other possible causes.

Treatment.—Where the damage is central and due to polio-encephalitis, nothing can be done beyond a transplantation of some other nerve into the facial muscle, the corner of the mouth being kept in good position by a simple hook. Where the damage is peripheral, that is with middle-ear disease or simple inflammation of the peripheral nerve, massage and electrical treatment will facilitate complete recovery.

THE MYOPATHIES (MUSCULAR DYSTROPHIES)

This heading embraces a group of diseases characterized by a tendency to weakness of the muscles

and sometimes wasting or replacement of the muscles by fat or fibrous tissue. This group is both familial and hereditary, the disease on the whole being passed down through the females to the males, but not necessarily all the members of a family are affected. Any muscle or group of muscles may be involved. The commonest types are the pseudo-hypertrophic type, to which the great majority of cases belong, the juvenile (Erb) type and the facio-scapulo-humeral type.

Pseudo-Hypertrophic Muscular Type

History and clinical picture.—Usually the subject is a boy, who is seen at the age of 4 to 6 years because he is not able to walk upstairs or get up from the floor in a normal manner. It appears that he was late in walking, but soon learned to run about more or less as other children. Then he gradually lost the power of mounting stairs or getting up from a reclining position. In rising from the floor he got up by rolling on to his abdomen, pressing himself up on his elbows, then getting on to his knees, then gradually climbing up his own thighs into the upright position (Figs. 46 and 47). On examining him it is noticed that the calves of his legs are much larger and apparently better developed than normal, but that the shoulder, pectoral and scapular muscles are very deficient or almost absent (Fig. 48). In attempting to lift such a child from the floor under the arms he almost slips through the hands because of this deficiency of muscle in the shoulder girdle. The arms are raised above the head with difficulty. The child walks in a characteristic fashion with marked lordosis and an apparent difficulty in getting the heel to the ground. There is a shortening of the tendo Achillis, and to balance himself the child must lean well back. He has a waddling gait. In going upstairs he pulls himself up by the balusters. The knee-jerks are present at first but ultimately disappear. In the other types of wasting described other groups of muscles are involved, but there is no essential difference between

Fig. 46.—Case of pseudo-hypertrophic muscular paralysis: first and second stages of rising from the ground.

one group and another as regards pathology. The prognosis is uniformly bad in all cases, as the disease tends to progress. Of the etiology nothing is known.

Treatment.—There is no specific treatment, the general treatment being the most important con-

Fig. 47. Case of pseudo-hypertrophic muscular paralysis: third stage of rising from the ground.

sideration. Such children should be kept "on the move," as once they are taken off their legs for any reason there is a marked tendency for them to show less ability to get about afterwards. Massage, active and passive movements, radiant heat, artificial sunshine should all be tried. Tonics and changes of air

are also important. If any tendons become markedly shortened or deformities occur, tenotomy should be resorted to. Organotherapy and injections of fibrolysin and other muscle preparations have not proved successful in the author's hands.

Fig. 48.—Case of pseudo-hypertrophic muscular paralysis, aged 9½, showing muscular calves and winged scapulæ.

Lumbar and Cistern Puncture.

Lumbar Puncture

This should be considered a very simple measure, and, if properly performed, is absolutely without danger. If the patient is mentally alert and normal it is best at all ages to give some general anæsthetic such as ethyl chloride or gas and ether, or at least to freeze the skin over the spine by spraying with ethyl chloride.

Preparation of the patient.—The whole lumbar and sacral region is well iodined, especially up and over the crest of the ilium. The patient is placed on the side with the knees drawn up and the head bent forward towards the knees, so that the back is well curved. The nurse or

assistant grasps the patient behind the knees and at the nape of the neck, approximating the two hands. In this way the spines of the vertebræ are drawn as far apart as possible, allowing the needle to enter the spinal canal. The left hand is placed on the crest of the ilium, which has been iodined, with the thumb opposite the space between the third and fourth lumbar vertebræ, which is easily marked out. The needle is taken firmly in the right hand and the point pressed in between the spines of the vertebræ at this level in the mid-line. On passing from half to three-quarters of an inch in, a click is felt as the needle pierces the stretched spinal theca. The stilet of the needle is then withdrawn and the fluid flows freely. If the child is crying the fluid runs more freely than when he is quiescent, and if he is deeply anæsthetized there is very little if any increased pressure. If the fluid is flowing unduly slowly the patient should be tilted so that the needle points slightly downward. If the needle is pressed forward through the spinal theca, striking the anterior surface of the spinal canal, a plexus of veins there will be pierced, so that there is a tendency for blood to be present in the fluid withdrawn. It is best therefore, once the click has been felt, to withdraw the stilet at once and not push forward.

How much fluid may be safely withdrawn?—As soon as the flow of fluid is seen to be very slow, withdrawal should cease. In cases of meningitis, where there has been increased pressure, it is well to run off as much as will flow freely, thus relieving pressure. Except in the extremely rare cases of cerebellar tumour, any ill-effects are unknown in children. Lumbar puncture should be considered a perfectly safe proceeding for any physician to carry out.

Cistern Puncture

Preparation of the patient.—The back of the neck should be shaved well up to the external occipital protuberance and the skin well iodined. The child is placed on its side, the head bent forward with the chin

on the chest and firmly held there. It is well to give an anæsthetic to patients who are quite conscious or normal mentally, but in meningitis, when the child is drowsy, this may not be found necessary.

Technique.—With a well-cleansed left hand the uppermost palpable spine of the neck should be felt and the lumbar-puncture needle passed in upwards and forwards to the base of the skull. This should strike the skull posterior to the foramen magnum. The needle is now passed up in the direction of an imaginary line passing through the centres of the ears. Should the base of the skull again be encountered the needle is withdrawn and passed slightly farther forward once more. After possibly two or three attempts, the needle, being directed slightly farther forward each time, will pierce a strong ligament and enter the cistern. The penetration of this ligament is distinctly felt and no mistake need be made. In the hands of the average physician this proceeding involves extremely little danger, and it is now accepted as a routine measure in certain cases in many teaching and children's hospitals. The advantages of cistern puncture are that, where a puncture in the lumbar region has turned out to be dry, fluid is almost certain to be obtained at this site. In injecting serum also one is much nearer the site of the lesion than when the lumbar route is used.

CHAPTER XII

MENTAL DEFECT

MENTAL defect in childhood may be primary or congenital, that is, the child may be born with it, or it may be secondary, that is acquired during life.

Congenital mental defect.—The commonest forms of this are *mongolism*, *congenital hydrocephalus*, especially the obstructive type where the fluid is pent up inside the brain due to some malformation, *microcephaly*, where the brain is malformed and has failed to develop (Plate 13), the head being consequently unduly small, and *simple congenital amentia*, where the child is backward and the brain has failed to develop, the head remaining normal in size. A proportion of the microcephalic or simple mental defects show a gross upper motor neuron lesion, that is, they may show spastic diplegia and resemble in all respects Little's disease (*see* p. 40).

Secondary or acquired mental defect.—Among the most common forms of acquired mental defect are *birth injuries* (*see* Chap. II, p. 41), the child being injured during the process of birth, bleeding having taken place into the ventricles or brain substance, with consequent gross cerebral damage. *Encephalitis lethargica* tends to leave the brain permanently damaged, thus the child may be simple or fatuous, or even a gross idiot. After *postbasic meningitis* hydrocephalus is liable to develop with accompanying mental defect. *Syphilitic meningitis* is another common cause of hydrocephalus. *Cretinism* is a form of mental defect secondary to thyroid deficiency and falls into this group. *Epilepsy* is probably the commonest form of acquired mental

Plate 13.—Brain in a case of mental defect (on the right) and a normal brain.
The occipital region in the normal brain (on the left) shows well-marked development and projects beyond the cerebellum, while in the other brain there is no such development.

defect. With each convulsion there is a tendency for small petechial hæmorrhages to take place into the brain substance, and as these clear up there is softening, or there may be sclerosis, as a result.

CONGENITAL MENTAL DEFECT

Mongolism

This is a form of mental backwardness, having a characteristic clinical picture, the facies bearing some resemblance to that of the Mongolian races. For practical purposes mongolism does not occur more than once in a family, although a few instances are known of a second having occurred.

Etiology and pathology.—Nothing is definitely known as to the cause of this defect, although several theories have been advanced. It is possibly due to a weakening of the reproductive organs in either the mother or father of the child.[1] To support this theory it has been noted that the subject of mongolism is usually the child of an aged mother, who has already given birth to a number of children, or of a father whose health has been undermined by nerve strain or who has suffered from shell-shock. It is thought by some to be a congenital malformation analogous to hare-lip, cleft palate or club-foot, especially since 15 per cent. of mongols have an accompanying congenital cardiac defect. An examination of the brain in such cases shows the convolutions to be fewer in number and poorly developed. Microscopically nothing characteristic has been described.

Clinical picture.—In the case of an infant the clinical picture is characteristic almost from birth. The eyes are seen to be slanting (Fig. 49). The child is given to sucking the tongue, which tends to protrude from the mouth and is inclined to be slim and pointed at its tip, and in time shows furrows on the dorsum. The head is definitely undersized, all such cases being

[1] Dora M. Berry, "An Investigation of Fifty Cases of Mongolian Imbecility from the O.P.D., Hospital for Sick Children, Great Ormond Street," *Brit. Journ. Children's Dis.*, Vol. 21, 1924, p. 259.

slightly microcephalic. There is often a slight squint present, and the whole infant tends to have poor tone in its muscles. This hypotonia is best seen in the arms and legs, especially the hands. A nurse holding the child will remark that it lies in her arms like a bag of jelly, instead of with the firm, live feel of the normal infant. There is often a tendency to umbilical hernia.

Fig. 49.—Mongol aged 3½ years, showing typical slant eyes.

The hands are small, the fingers tapering, and the little finger especially is shortened and inclined to curve in. The skin is smooth, but tends to roughness in those parts which are exposed to the weather. The colour of the cheeks is high. Very often the child is said to be too good, seldom crying.

An older mongol presents many of the characteristics just described. The mouth turns down at the corners, and the head is definitely small. An examination of

the heart shows some congenital malformation in about 15 per cent. A considerable proportion of mongols have some other congenital malformation present. The child is undersized and the progress of growth is as a rule poor; he is slow in sitting up and walking. The mentality may be very deceptive, as the child seems bright, takes a great interest in its surroundings, smiles up into the face of its mother and is most affectionate. It is not until it is expected to talk that gross mental defect is suspected. Mongols are as a rule fond of music, affectionate, gentle in disposition, occasionally they are destructive. Imitation is highly developed, initiative is lacking. They can be taught to be clean in their habits and moderately obedient, but they cannot be trusted to any great extent and do not prove to be useful members of society. Institutional treatment has a beneficial effect. Because of the short nasopharynx the child tends to make a snorting noise as if adenoids were present, almost from birth. Frequently adenoids and enlarged tonsils do appear later, and should be dealt with.

Prognosis.—Most mongols die before their twentieth year.

Treatment.—There is no rational treatment for this condition. The giving of thyroid has absolutely no effect on the mentality of these children, but since there is a natural tendency to improve, thyroid has gained an unwarranted reputation in this connexion. Most help is gained by concentrating on the education of the child and keeping up its general health. Careful mothering, the inculcation of cleanliness and the checking of objectionable habits, are most essential. Great help can be obtained from an instructive booklet by Dr. John Thomson, entitled "Opening Doors" (Oliver and Boyd). Since such a child is prone to the development of catarrhal infections, rickets and other diseases, greater care of the diet, and attention to tonsils and septic teeth, are required. Solid food is not taken well, largely because of the child's mental condition, and fluid nourishment should be restricted, so as to

encourage him to take his solids. Cod-liver oil is indicated.

Microcephaly

This is a form of congenital amentia, the brain being under-developed and the circumference of the head less than normal (*see* p. 7). When present in a gross

Fig. 50.—Microcephaly.

degree there is no difficulty about the diagnosis. The forehead recedes, the head sloping to a point from all sides and rising sharply towards the vertex (Fig. 50). There are, however, many mild degrees of microcephaly, where the head is merely slightly undersized. Mongolism is in reality a mild degree of this condition. Very occasionally more than one microcephalic occurs in a family. The etiology is unknown.

Pathology.—There is general aplasia of the brain substance, especially of the posterior portion of the brain along the cerebellum, which is often markedly exposed. The various layers of nerve cells in the cortex are undeveloped. The fontanelle tends to be closed at a very early age in these cases and the head assumes its characteristic shape from birth onwards.

Prognosis.—Since convulsions are common in this condition and the children are prone to diseases such as tuberculosis and have a poor immunity to infections, they have only a short span of life.

Treatment.—There is no rational treatment. Education and attention to the general health, with institutional life, are indicated.

Hydrocephalus (Water on the Brain)

An enlargement of the head due to an increase of cerebro-spinal fluid either within the brain (internal hydrocephalus) or outside the brain (external hydrocephalus).

Etiology and pathology. The non-communicating form.—In this type the fluid is pent up inside the brain and is unable to escape into the subarachnoid space and spinal meninges, where it would be absorbed and find its way back into the bloodstream. This may be due to developmental errors, such as a congenital closure of the foramen of Monro or the aqueduct of Sylvius. Basal inflammations, such as occur in post-basic meningitis due to meningococcal infection or to syphilis, close the foramina of Magendie and Lushka and are responsible for most cases of this form of hydrocephalus. Tumours pressing on and closing the various foramina or on the fourth ventricle also cause internal hydrocephalus. Hæmorrhage into the ventricle may occur at birth, blocking the foramina or the aqueduct of Sylvius and thus producing hydrocephalus. This is most common in premature infants, and such a causation may account for the history of a normal labour with a normal-sized head which rapidly increases in size after birth.

Communicating form.—Although in this form the fluid may pass freely from the choroid plexus to the subarachnoid space, the mechanism for absorbing the fluid and carrying it away may be defective. This is sometimes due to a congenital malformation, but is much more often acquired as a sequel of inflammation. The delicate stomata of the lymphatics have been closed

Fig. 51.—Hydrocephalus. (*By courtesy of Dr. G. F. Still.*)

or occluded as a result of acute or chronic infection. Mild forms of meningitis, such as those due to the meningococcus or to syphilis, are most often responsible.

Again, the absorption mechanism may be perfect, but too great a quantity of cerebro-spinal fluid may be excreted by the choroid plexus. This may be congenital, due to some endocrine disturbance or other cause at present unknown.

From long-continued pressure the brain substance is reduced to extreme thinness. At autopsy in some

cases it is as thin as a sheet of paper and does not bear handling. The motor area and other portions are thus partially or entirely destroyed, so that mental defect and spastic paralysis are to be expected. The pituitary fossa and the pituitary gland itself are atrophied.

Clinical picture.—Labour may be found difficult due to the enlarged head of the infant, very often

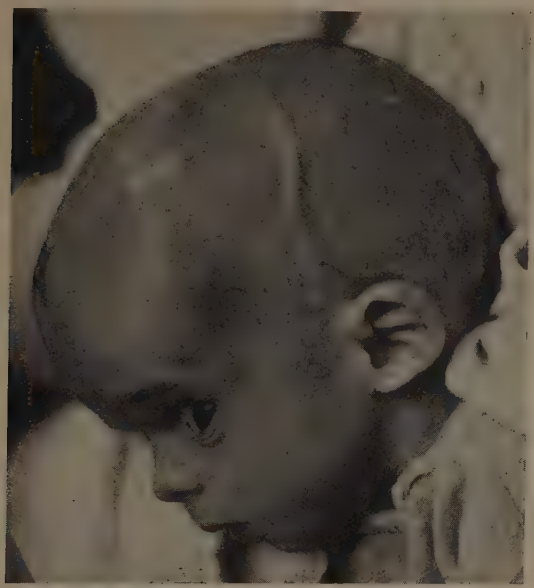

Fig. 52.—Hydrocephalus. (*By courtesy of Dr. G. F. Still.*)

requiring its destruction. These cases are, of course, congenital. On the other hand the infant may be born apparently normal and in the first few weeks of life the head may increase extremely rapidly, so that by the age of 3 to 6 months it has attained enormous proportions. The forehead is very prominent, overhanging the rest of the face (Figs. 51, 52 and 53), the eyes being everted and the sclerotics showing above. The fontanelle

is widely open, sometimes being 3 to 5 inches across. The sutures are also widely open, and the posterior fontanelle fails to close. The bones of the skull are seen to be greatly thinned and on pressure may yield slightly. A powerful lamp placed near the head shows it to be partially translucent. When hydrocephalus develops after postbasic meningitis in an older child, however, such an extreme picture does not occur. In the infant, who tends to lie where it is placed, in addition to protrusion downward of the eyes, constant lateral nystagmus is a frequent symptom. A tendency to vomit and thus soil the pillow is a great danger, as the head is then liable to develop cellulitis, which heals badly. Should the hydrocephalus be a mild one, a balance is struck between the fluid excreted by the choroid plexus and that carried away by the lymphatics. Thus the head ceases to enlarge. The small body in time gradually " catches up " in growth to the huge head and ultimately very

Fig. 53. Hydrocephalus following postbasic meningitis showing marked wasting and overhanging brow. (*By courtesy of Dr. G. F. Still.*)

little if any enlargement is to be noted. Due to the thinning of the brain itself and the constant pressure on the pituitary and the fourth ventricle, the clinical picture may vary greatly. Thus signs of an upper motor neuron lesion may be present, and spasticity, especially of the legs, is common. There is usually also a tendency to put on much flesh, probably due to pituitary damage —the "fat child."

Mentality.—Many cases show very slight mental defect, but the majority are grossly affected, and all show emotional instability, tempers, tears or fits of stubbornness being common. Fifty per cent. of all hydrocephalics suffer from convulsions, and this serious complication tends to shorten their lives.

Diagnosis.—In studying a case of hydrocephalus it is first necessary to ascertain whether it is of the communicating or non-communicating type. A lumbar puncture or cistern puncture is useful, as if the fluid flows freely and a considerable amount may be withdrawn, it is safe to assume that this is the communicating type. Various dyes have been employed to prove this. For example, 1 c.c. of methyl violet solution is injected by means of a very fine needle through the open fontanelle into the ventricle; 15 or 20 minutes later a lumbar puncture should be done and the dye should be recovered in this fluid. If the fluid flows badly from the lumbar region or if the dye fails to appear it may be assumed that it is a non-communicating case, that is, there is a block somewhere at the foramen of Monro, aqueduct of Sylvius, foramen of Magendie, etc.

Intraventricular indicator.— Neutral phenol-sulphone-phthalein (Dandy) (prepared by W. Martindale, 12, New Cavendish Street, W. 1), is used. The lateral ventricle is punctured and 1 c.c. of the solution in the syringe is mixed with the cerebro-spinal fluid by the withdrawal of 2 c.c. into the syringe barrel. This mixture is then injected into the ventricle. Lumbar puncture is performed 5 minutes and 30 minutes after the injection, and the cerebro-spinal fluid is run into a test-tube with a few drops of 25-per-cent. sodium hydrate.

Recovery of the phenol-sulphone-phthalein shows that the hydrocephalus is extraventricular or communicating. Non-recovery shows that it is intraventricular or non-communicating.

Injection of air (pneumo-radiogram).—For some time these cases have been investigated by withdrawing the cerebro-spinal fluid and allowing air to pass up the spinal canal and replace the fluid both within and without the brain. X-ray photographs then show the exact state of affairs (Plate 14). Most valuable information can be obtained in this way, and with careful manipulation there is extremely little danger of any fatality. The air should, of course, be sterile before it is injected. As a rule, within 24 hours it has been absorbed. In the closed or non-communicating form the air must be injected directly into the ventricle, and on this account the method described is much less suitable as a means of investigation. The needle passing through the brain substance unavoidably damages it, and there is a tendency for bleeding to occur. With most skilful technique, however, the attempt should meet with success, but it is not recommended otherwise.

Treatment.—Before any form of treatment is thought of, the Wassermann reaction should be resorted to in all cases, as hydrocephalus may be the result of syphilitic meningitis. Such cases show the best response to treatment and the results are excellent. In the communicating forms no rational treatment is available, but in the non-communicating forms various operations have been devised to enable the pent-up fluid to escape into the subarachnoid space, where it can be successfully dealt with. For a description of these measures, *see* "Surgery in Childhood," by John Fraser. Various forms of medical treatment have been suggested. Diuretin given by the mouth and causing an increased flow of urine is said to decrease the intracranial pressure. The author has not found this. Purges such as magnesium sulphate given by the mouth are not a suitable form of treatment for infants and children. Intravenous hypertonic saline may have some transitory effect, but it is

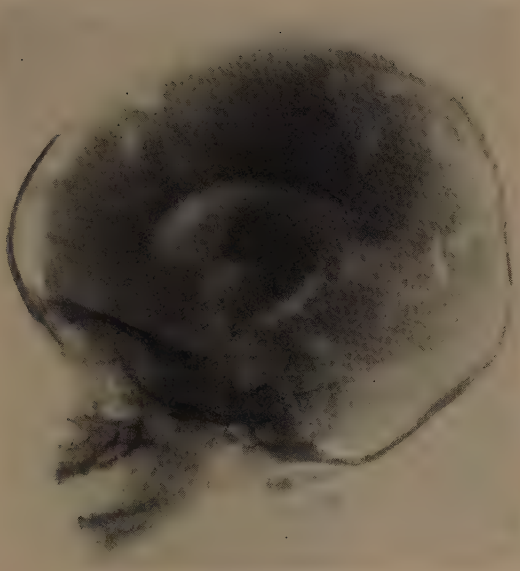

Fig. 1.—Lateral ventricle.

Fig. 2.—Front view showing both lateral ventricles.

Plate 14.—Pneumoradiograms showing air in the ventricles of the brain.

not a form of treatment which can be administered repeatedly.

Fig. 54. Showing state of thyroid after death in case of cretinism.

A, Larynx with no trace of thyroid.
B, Larynx of a normal child, showing well-marked thyroid.

SECONDARY OR ACQUIRED MENTAL DEFECT

Cretinism

This condition may be present from birth or acquired at some later date. In the British Isles as a rule it is congenital.

CONGENITAL CRETINISM

Diagnosis is possible at the age of 3 to 4 months. The sex distribution is equal.

Etiology and pathology.—No explanation has

Fig. 55.—An untreated cretin aged 5 years, showing the large abdomen with umbilical hernia, protruding tongue, and pads of fat on the shoulders.

been offered of the congenital absence of the thyroid gland, and it may be looked on as a malformation, the absence in most cases being complete (Fig. 54).

Absence of the thyroid gland produces a typical clinical picture and certain pathological sequelæ throughout the body. An excess of fat is laid down in the subcutaneous tissues, especially about the shoulders, just as in the hibernating bear. Fat is also laid down in many other parts of the body, such as the tongue and the subcutaneous tissues. The mentality is backward.

Clinical picture.—The cretin is pale and has a yellow-tinged, harsh skin. The *hair* is straight and dry; the *voice* is hoarse and low-pitched; the *tongue* is greatly enlarged, and pro-

trudes from the mouth continuously, since it is actually too large for the mouth to contain (Figs. 55 and 56). The body is firm and stiff, and inclined to be muscular. The child is *slow in all his movements*, the smile comes and goes slowly and his *mentality* is greatly retarded. He is very backward in learning to walk, and may be several years old before he can accomplish this if untreated. He is inclined to make *snorting noises* with his nose. The *temperature* is subnormal, the hands and feet being cold and the pulse slow. He is chronically constipated. The growth of the *skeleton* is slow, and he is *dwarfed* in size. His *teeth* are late in appearing and when they do appear, tend to be carious. The *thyroid gland* cannot be felt, but it is seldom that cretinism is accompanied by other forms of congenital malformation.

Very often mistakes are made in diagnosis between cretinism and mongolism. There ought to be no difficulty whatever, as the following summary will show:

Cretin	Mongol
Pale, yellowish skin.	Pink-cheeked and fresh looking.
Skin harsh.	Skin soft.
Hair straight and dry.	Hair quite normal.
Tongue greatly enlarged and protruding continuously.	Tongue small, pointed at the tip, protruded at times with a sucking movement.
Voice harsh and low-pitched.	Normal cry.
Body firm and stiff, inclined to be muscular.	Body soft, hypotonic, and flabby.
Movements slow.	Movements quick.
Backward in learning to walk.	Normal or only slightly backward.
Snorting noises in nose.	Snorting noises in nose.
Temperature subnormal.	Temperature normal.
Chronically constipated.	Not necessarily constipated.
Growth of skeleton slow, dwarfed in size, teething backward.	Not necessarily under-sized; with proper diet teething normal.
Eyes normal.	Eyes slanting and almond-shaped.
Thyroid gland cannot be felt.	Thyroid gland palpable.
Circumference of head normal.	Greater or less degree of microcephaly.
Seldom accompanied by congenital malformations.	Heart disease or some other malformation often present.

Prognosis.—In cretinism this is a matter of great difficulty, as it depends entirely on how early and how efficiently the child is treated. There are, however, certain cases, few in number, in which there is mental deficiency despite treatment with thyroid. The majority of cretins are treated late and inefficiently and therefore lag some distance behind the normal child, failing to catch up or reach the normal standard. A small proportion taken in hand early and properly treated become apparently absolutely normal.

Fig. 56.—An untreated cretin aged 8 months. Note the thick protruding tongue.

Treatment.—Thyroid gland must be administered, beginning with small appropriate doses. One brand should be used and adhered to by the physician, as there is a great variation in the strength of the different preparations. The author uses Thyroid Gland (Burroughs, Wellcome & Co.) and, for an infant a few months old, begins with a dose of $\frac{1}{10}$ grain three times daily, slowly increasing this week by week until the greatest dose is reached which the infant can tolerate without diarrhœa and without undue loss of weight. This is usually about a total of $\frac{1}{2}$–$\frac{3}{4}$ grain in the day. The signs of recovery are that the constipation ceases, the bowels becoming normal, the cold hands become warm, the dry hair soft and even curly,

the increased subcutaneous fat disappears, the skin is soft and loses its yellow tinge, becoming pink and white. The voice ceases to be hoarse and is clear and infantile. The tongue is small and does not protrude from the mouth, the mentality is quickened, and the child smiles and obviously appreciates its surroundings (Fig. 58).

Signs of too much thyroid.—When too much thyroid is being administered there is a tendency to undue loss of weight, looseness of the bowels and an elevated temperature. The child is nervous and fearful, being too timid to cross the street and afraid of cats and dogs. Finally there are attacks of palpitation, with sudden fainting fits, probably accompanying the palpitation. An average cretin a year old will tolerate about $1\frac{1}{2}$ grains in the day, and between one and three or four years appears to tolerate 2 to 3 grains per day. After that the dose remains stationary. Each child, however, is a law unto itself, and only by very careful observation can the proper dose be ascertained. Recently the dose of thyroid has been controlled by metabolic estimations.[1] During the winter months the

Fig. 57.–Cretin before treatment.

[1] H. Gardiner-Hill, P. C. Brett and J. Forest Smith, "Carbohydrate Tolerance in Myxœdema," *Quart. Journ. Med.*, Vol. 18, No. 71, April 1925, pp. 327–334.

dose of thyroid is a larger one than during the summer. For instance, a child who tolerates 3 grains during the winter months will tolerate not more than 2 grains during July and August.

After the administration of thyroid there is a rapid mental and physical development and a greatly increased growth of the bones, which brings in its train the danger of rickets. It is essential, then, that the diet should be carefully investigated and adjusted, and that cod-liver oil should be administered at all times along with the thyroid.

Fig. 58.—Cretin after several years' thyroid treatment.

ACQUIRED CRETINISM

Occasionally an infant is born normal and in the first few years of life has some acute illness or local inflammation in the region of the thyroid. Typical myxœdema follows and the picture of cretinism results. Such cases react rapidly to treatment with thyroid. *Adenoma* of the thyroid, or benign goitre, is very rare in the British Isles.

Epilepsy

The term epilepsy is a most misleading one, and should, in our present state of knowledge, be limited to

those cases, with recurring attacks of either minor or major fits, for which no explanation can be given.

Age-incidence.—Epilepsy may appear at any age from early infancy to late adult life. True epilepsy, however, is uncommon before the first dentition is complete, that is, between the ages of 2 and 3 years. Fits or convulsions at an earlier age than this are usually due to birth injury or are of rachitic origin (spasmophilia).

Etiology and pathology.—With the exclusion of cerebral tumour, cerebral hæmorrhage, developmental malformations and inflammatory lesions of the brain accompanied by attacks conforming to a certain clinical picture, we are left with so-called *idiopathic epilepsy*. Epilepsy, as far as we know at present, is due to a sudden explosive liberation of nervous energy in certain motor cells, the cause of this liberation being quite unknown. The condition is associated with a most unstable nervous system. The determining factor in precipitating a fit in certain cases would appear to be some error in metabolism, as accompanying or preceding the attacks constipation and acute digestive upsets are common.

With each recurrent fit there is cerebral engorgement resulting in minute petechial hæmorrhages. With the clearing up of these hæmorrhages sclerosis and softening, with mental deterioration, are inevitable. The fits must be stopped if the child is to remain mentally normal.

Clinical picture.—In what has been considered a perfectly normal child there may suddenly occur a convulsion (*grand mal*). This may be preceded by a cry, then the child falls and tonic contractions of the muscles take place lasting perhaps only for a moment, or for several minutes. Now clonic spasms of the muscles take place, the whole child working, and froth appearing at the mouth. The bladder is emptied and often the bowel also. After a period varying from a minute or two to several hours on occasions, the spasms pass off and the child sinks into a deep sleep. There is great variability in the length of the different periods,

and the whole fit from start to finish may occupy only 30 seconds.

The attacks may be very slight (so-called *petit mal*). In such an attack there is only a short pause or little "absent moment" (Fig. 59). The eyes are jerked or twitched in one or other quadrant, or there may be twitching of the facial muscles or shoulder along with this absent expression of the eyes, the whole thing being over in a second or two. Petit mal attacks often occur many times daily.

Salaam type of petit mal.—This is most often seen in the infant of a year or more who has learnt to sit on its mother's knee. It will suddenly bend forward, throwing the arms from its sides with the hands clenched, then straighten up, the fit being over. Gross mental deterioration is common where there are frequent petit mal attacks.

Diagnosis.—When the fits, either of grand mal or of petit mal, are typical, the diagnosis is rarely doubtful. After the age of two, when rickets becomes extremely uncommon, spasmophilia as a cause of fits may be more or less excluded. It is unsafe to diagnose epilepsy under the age of two without the most careful and prolonged observations. The disease, however, very often commences in the first two years of life, and this fact should be realized more widely. Since fits are more common during the night than during the day, they may have been present in many cases for some time before their existence becomes known. Petit mal attacks are often present for years without being recognized, although in some cases the fact that the child is abnormal mentally becomes increasingly apparent to the parents. Epileptics of long standing seem to lose all sense of awe or fear, they make friends too easily and have no power of concentration. Thus, the child epileptic plays for a moment only with a toy before discarding it. The fits occur in bouts or groups, many at a time, and then for no apparent reason there is a complete cessation, followed by another group. The explanation of this grouping of the fits is not forthcoming.

Fig. 59. – Case of epilepsy in a girl aged 8, having repeated petit mal attacks. The lower photograph shows her in a typical attack two minutes after the upper one was taken.

Differential diagnosis.—Shortly after birth, cerebral hæmorrhage must be excluded. In regard to children up to 2 years of age, rickets should be borne in mind. At any age fits may be a symptom of an acute inflammation of the brain, such as encephalitis, poliomyelitis or meningitis. Of the more chronic infections, syphilitic meningitis, giving rise to hydrocephalus, is important. In older children a brain tumour may simulate epilepsy, while congenital malformation of the brain, cerebral degeneration or pyknolepsy may be the cause of fits. *In every case of epilepsy a Wassermann reaction and a thorough examination of the eyes should be carried out as a routine, in addition to a systematic examination of the nervous system.*

In epilepsy, as in all diseases, there must be a beginning, and the first fit, if it be grand mal, is bound to give rise to a doubt as to its true nature. Probably among the most puzzling and difficult cases are those where a child has a major seizure, epileptiform in character, accompanying each bout of fever. Thus at the onset of pneumonia or measles, or even in acute tonsillitis with high fever, the child lapses into status epilepticus, such attacks recurring at intervals of a few months, but passing off ultimately at the age of five or six. There is, however, no mental retardation. An explanation of such convulsions is not known, but in a certain proportion of these cases the cerebro-spinal fluid will be found to contain much acetone.[1] Breath-holding convulsions occurring between the age of one and two years, also simulate epilepsy closely. For a description of these convulsions *see* p. 349.

Prognosis.—The immediate outlook is excellent, the mortality from epilepsy by itself being slight. The ultimate prognosis, however, cannot be regarded as anything but unsatisfactory. In slight cases years may elapse without a recurrence; in fact, there may never be more than the one bout of fits. In other cases, however—and it is impossible to detect these—frequent recurrences may take place, with definite progressive

[1] Donald Paterson and G. W. Bray, "Infections as a Source of Recurrent Epileptiform Seizures in Children," *Arch. Dis. Child.*, Vol. 4, No. 27.

mental deterioration. Intercurrent disease claims the greatest proportion of such children, largely because they are confined and cannot get about. Except, perhaps, in a few cases, an accurate prognosis is not possible in our present state of knowledge.

Treatment of an attack.—In the short momentary attacks, or in those lasting a few minutes only, the whole thing is as a rule over before any treatment can be administered, beyond preventing the child from biting his tongue or bruising himself. In attacks lasting more than a few minutes the treatment is that of status epilepticus, to be dealt with later.

General treatment. 1. *Dietetic.*—In America epilepsy is said to have been treated successfully in a proportion of cases by putting the child on a ketogenic or acidosis-producing diet. It has been observed that during pregnancy, febrile illnesses, or periods of fasting or starvation, a condition of acetonæmia obtains and that in some cases fits are held in check at these periods. On the assumption that the presence of the acetone is the inhibiting factor, acetonæmia has been artificially produced by means of a high-fat, low-carbohydrate diet, and Moriarty [1] and others claim good results.

McQuarrie [2] claims that the whole question turns on the water balance or retentivity of the tissues. He claims that the ketogenic or acidosis-producing diet causes a negative or poor retention of fluids in the tissues, while a diet high in carbohydrate has a reverse effect. He recommends a high fat, low carbohydrate diet and restriction of the fluid intake.

Diet for an epileptic child of 5 to 7 years.—The diet should be one high in fat, with a restricted carbohydrate and fluid intake. A moderate amount of protein is allowed. Thus, at breakfast, the child should have eggs or fat bacon, a very little creamy milk to drink and a small portion of bread spread thickly with butter.

[1] M. E. Moriarty, "Epilepsy: Chemical Investigations of Rational Treatment by Production of Ketosis," *Amer. Journ. Dis. Child.*, Vol. 33, p. 218.

[2] Irvine McQuarrie, "Epilepsy in Children: the Relationship of Water Balance to the Occurrence of Seizures," *Amer. Journ. Dis. Child.*, Vol. 38, pp. 451-467.

At midday, fried fish, fat pork or ham or other fat meat should be given, with a very small quantity of potato and plenty of green vegetables. Plenty of butter should be served with the latter. As a second course cream-cheese and celery may be given, or a quarter of a sliced orange or half an over-ripe banana with two or three tablespoonfuls of thick cream. For tea-supper the child should have 3 oz. of creamy milk, egg or cream-cheese sandwiches, or a small portion of bread with clotted cream.

The proportion of fats must be adjusted according to the presence or absence of acetone in the urine. If fats are pressed too far vomiting occurs, and the diet must then be stopped, very little fat given and carbohydrate offered freely.

Restriction of drinks.—The diet should be as dry as possible, not more than three half-glasses of fluid of any sort being allowed during the day. *Excellent results have been obtained by the author with the few cases on which this treatment has been tried.*

2. *Medicinal.*—For many years *bromides* have been employed, and there are various patent medicines on the market, for the most part containing bromide as the basis. Combinations of the bromides of ammonium, potassium and sodium are said to be better than any one singly. *Luminal sodium*, combined with potassium bromide and made up with a mild antiseptic such as chloroform-water (otherwise such mixtures do not keep), has been most successful in the author's hands.[1] A suitable mixture for a child of 5 to 10 years would be: luminal sodium, ½ grain; potassium bromide, 5 grains; chloroform-water to 2 drachms, to be given night and morning. Tincture of belladonna, 5 to 10 minims, can also be added to this mixture with benefit in some cases which otherwise do not respond. Toxic effects and rashes, mental deterioration and habit have been said to develop with prolonged use of luminal, but the author has observed no ill-effects to follow its

[1] J. Tylor Fox, "Luminal Sodium in the Treatment of Epilepsy," *Lancet*, Sept. 17, 1927, p. 589.

employment in some hundreds of cases. In a considerable proportion of cases the fits are completely held in check by this treatment.

3. *General management.*[1]—There is no condition in which the general management of the child is more difficult to arrange or is more important. As a rule epilepsy excludes the child from school, unless it be some small private school among friends, or some special institution. Naturally autocratic and difficult to control, he becomes doubly so as constant care and watching make him self-centred and introspective. Unable to concentrate for any length of time on one subject, he is difficult to amuse or teach. A capable nurse or governess who understands the situation should be secured wherever possible, and the child must be skilfully disciplined and checked, so that tears and bursts of temper are avoided. His play should be systematic, not spasmodic, and his day should be laid out so that every available moment is occupied definitely and not haphazard. A rest after the midday meal is beneficial. Romping with playmates is said to excite the epileptic child unduly, but the good effects of companionship and the greater ease with which he may be disciplined must be carefully considered before he is cut off from his brothers and sisters or other children. Dr. John Thomson quotes Dr. Ashby as saying: "The child is a social animal, and the society of other children, if well under control, has a powerful influence for good; and the more neurotic the child, the more need there is for his contact with others. To allow the neurotic child to feel that he is the first to be considered, to let him think that every little ache, every little disappointment, and every discomfort are sore trials, is to be doing all that can be done to blight his future. Give him daily employment, everything well within his powers, and look for accurate performance. Defer if you will all abstract learning to a later day, but find for him an active and occupied life;

[1] J. Tylor Fox, "The After-care of Epileptic Children," *Lancet*, Sept. 15, 1928, p. 545.

lend him a firm hand that may help to forge and strengthen the links of voluntary power and self-control which will prove important factors in preventive medicine and save many a sorrow."[1] With all this the author entirely agrees. A visit to the Epileptic Colony at Lingfield by anyone specially interested in the subject will show what great benefit there is to be gained by institutional life in these cases.

Status Epilepticus

Children subject to fits, either with or without an organic brain lesion, react violently to acute infections. For instance, a child with spastic paralysis (Little's disease) having epileptic fits at intervals may pass into a status epilepticus very easily with an attack of influenza or acute tonsillitis. In this state the child may lie in a stuperose condition for some hours or even days, with almost continuous convulsions, the tonic stage giving place to the clonic, and that again reverting to the tonic, with very little remission between. Swallowing is impossible and death results if this state continues for any length of time. In most cases there has been some cerebral lesion previous to the onset.

Treatment.—A lumbar puncture is of the greatest value and should be performed first in all cases. The relief of intracranial pressure is very effective in stopping the fits. Chloroform or ether is efficient in stopping them for the time being, and chloral, bromide, chloretone or paraldehyde per rectum are also helpful. Rectal saline-and-glucose or glucose by the mouth is indicated as early as possible.

Pyknolepsy[2]

The features of the recently described condition of pyknolepsy are:

1. The absence of mental degeneration.

[1] John Thomson, "Clinical Study and Treatment of Sick Children" (Oliver and Boyd), pp. 603–4.

[2] W. J. Adie, "Pyknolepsy," *Proc. Roy. Soc. Med.* (Neurol. Section), Mar. 1924, Vol. 17, No. 5, p. 49.

2. The failure of anti-epileptic treatment to affect the frequency of the fits.

3. The occurrence of a very large number of fits each day, 80 to 100 at least.

4. The sudden onset and sudden cessation of such fits. Cessation takes place after a period of from one to three years, and the fits never return.

Convulsions in Infants.

The diagnosis of a convulsion is often difficult because of the necessity of getting a clear and satisfactory history or description from the mother. Attacks of wind, screaming, or even masturbation may be confused in the mother's mind with a convulsion. Generally speaking "convulsions" are like "worms"—they must often be seen to be believed.

A convulsion or fit is usually ushered in by a general stiffness of the whole body. The eyes are fixed and are usually widely open, and the arms are stretched out with the fingers clenched. This stiffness may last for a few moments or even for several hours. Foaming at the mouth is often a feature at this stage. During the "stiff" period the child assumes a bad colour, becoming either blue or extremely pale. Following this stage there are movements of the limbs and whole body (so-called clonic stage). The fit then passes off and the child relaxes and may pass into a deep sleep. He may or may not have passed urine or fæces.

If the fit is a slight one the whole thing may be over in a few moments, with only the dropping of the head and a dazed look in the eyes marking a minor convulsion.

Predisposing causes of convulsions.—Undoubtedly among the members of some families there is an *error or defect in the physical or mental make-up* which makes them especially liable to convulsions. By far the commonest predisposing cause in infants is *rickets*. Here with a low blood-calcium the nervous system becomes unduly excitable and such minor disturbances as teething, indigestion or peripheral

stimuli such as worms will often excite a convulsion. A fit of temper, a fall or shock may easily bring one on, and slight feverish turns or infections in this type of child often precipitate an attack of convulsions.

Among other predisposing causes is, undoubtedly, *congenital malformation of the brain* or *cerebral damage at or following birth.* That is, all cases of primary and secondary amentia, which have been described previously in this chapter, are especially liable to convulsions.

Infections or feverish turns or digestive upsets may act as precipitating causes of convulsions. In certain children any hyperpyrexia is accompanied by convulsions. This state of affairs usually begins between the ages of $1\frac{1}{2}$ and 2 and continues up to the age of 5 or 6 or even later. At intervals of three or four months or longer, the child has a series of major convulsions, invariably accompanied by fever, and a close examination will show that some infection has taken place, as a rule in the nasopharynx (tonsils). After a few moments or, possibly, hours, the convulsions cease, and the child appears to recover completely. Very often vomiting may have preceded this turn and may persist afterwards with occasional pale motions. The attacks are described by the mother as feverish bilious attacks, and that accurately describes the clinical picture. Such cases recover completely, in the author's opinion, on removal of the tonsils or source of infection, provided the child is put on an anti-ketogenic (antibilious) diet, low in fats and high in carbohydrate (sugar).

Age must be considered an important factor in predisposing to convulsions, as a sudden pain or shock which, in an older child, would affect only one part of the body and reach only one part of the brain, in the infant, with its poorly insulated nervous system, may affect the whole system, and result in a general convulsion.

If the fits come on immediately after birth they are unlikely to be due to rickets, and are most probably due to some birth injury, but if they appear after the age of 14 days, then birth injury as a cause is unlikely.

Convulsions in whooping-cough.—These are dealt with elsewhere, but undoubtedly cerebral congestion and asphyxia, which occur during a spasm of whooping-cough, will produce a definite convulsion in some children. The outlook is extremely grave in such cases.

Breath-holding convulsions.—Among the most interesting causes of convulsions is the habit of " breath-holding " which prevails among certain children, usually between the ages of 1 and 3 years. As a rule either they are " only " children or they have had an undue amount of attention. Possibly the organic basis underlying these attacks may originally have been laryngismus stridulus or a laryngeal spasm in rickets, but all the cases seen by the author have continued past the rachitic age and have shown no signs of active rickets. In such cases a child about the toddling age falls, or is crossed in some way, causing a burst of crying. It may be placing him on his chamber or taking something out of his hand which evokes this outburst. After a few cries the breath is held, the face becomes purple, the eyes protrude and are congested, then the child, by this time quite stiff, passes into a stage of clonus, and finally relaxes, becoming perfectly normal and resuming his play within the next two or three minutes. Convulsions of this kind may occur many times daily and are most alarming. Sedatives do not appear to be of much value. Cure of the convulsions is largely a matter of management ; thus, if the child can be made to take a deep inspiration at the onset of the attack, the convulsion is aborted. A sponge dipped in cold water and applied to the face or chest causes the necessary inspiration. The child must be taught discipline and self-control. All the cases seen by the author have quite recovered, and there is no tendency to epilepsy or mental trouble afterwards. The usual time of recovery is about the age of three.

Convulsions with dehydration.—Another group subject to convulsions are infants who develop the condition during an attack of acute diarrhœa with

marked dehydration. Whether the cause is undue concentration of the blood and a resultant concentration of the urea and other solid constituents of the blood, ultimately reaching a point where convulsions occur, cannot be said. There appears, however, to be a direct relationship between dehydration and the convulsion. After such an operation on an infant as that for congenital pyloric stenosis, when an adequate diet is being very slowly introduced, there is a tendency for convulsions to occur, possibly due to dehydration or to a very low blood-sugar, the consequences of an inadequate diet.

Convulsions due to hyperpyrexia.—In conditions of septic absorption such as eczema or after burns, there is a tendency for the temperature to rise rapidly and for convulsions to supervene. Convulsions may also occur with the pyrexia of pneumonia and pyelitis.

Spasmophilia.—Undoubtedly the commonest cause of convulsions in the first two years of life is spasmophilia or nervous rickets, a condition characterized by a low blood-calcium which causes undue excitability of the nervous system. This is shown by tapping over the facial nerve, when a contraction of the facial muscles occurs (Chvostek's sign). A child in this excitable state may, from very slight causes, have a convulsion. The treatment in such cases is simple and most effective. The patient should be put on a proper diet, and if there is a deficiency in curd (calcium) it should be made good. Cod-liver oil should be administered in some form such as emulsion, a teaspoonful three times daily. A little chloral should be given, say half a grain for a very young infant to a grain for an infant a few months old, three times daily. The specific treatment, however, is the administration of calcium chloride. This should be given in such a mixture as calcium chloride 3 grains, syrup q.s., aqua to 1 drachm, four times daily for three days, then three times daily. The child should be given artificial sunlight or natural sun-baths to raise his blood-calcium, and within a few hours the fits should cease, the tendency to convulsions clearing up within a few days. With calcium chloride there may be a

tendency to looseness if the dose is too large. The dose recommended is comparatively a cautious one, and considerably larger quantities are tolerated by many children with good results.

Uræmic convulsions.—Uræmic convulsions are extremely rare in infants. A child with syphilitic nephritis may show such convulsions along with marked œdema, but this is a rare condition. Again, they may occur in children suffering from polycystic disease of the kidney or some other abnormality of the urinary tract.

Prognosis.—This, of course, largely depends on the cause of the fits. The first duty of the physician is to stop the fits, as the longer they continue the worse the ultimate outlook. With each convulsion there is a tendency for slight but permanent cerebral damage.

Treatment of convulsions.—The child should be placed in a mustard bath, or warm water if mustard is not available. This measure will be found to help to relax the stiff muscles and spasms. If able to swallow, the child should be given some sedative drug such as chloral. New-born infants should be given a half-grain dose at two- or three-hour intervals until the convulsions cease. After the age of three months grain doses can be given at the same intervals. If the child is unable to swallow, the drug must be given by the lower bowel, the rectal dose being about four times the amount given orally. Inhalation of chloroform should be resorted to with prolonged convulsions. Lumbar puncture is a valuable therapeutic measure, and if possible should always be carried out. Where the convulsions are thought to be due to an infective acidosis the administration of saline and glucose rectally is indicated. For the treatment of status epilepticus *see* p. 346.

CHAPTER XIII

FUNCTIONAL NERVOUS DISEASES

THE NERVOUS CHILD

Much has been written in recent years about the nervous child and his peculiarities, and there is often a tendency to go to the extremes of attributing all his nervous faults either to organic disease on the one hand, or to faulty management on the other. A nice balance, however, must be maintained between these two. In endeavouring to ascertain whether the fault lies in the management or environment, or is to be attributed to organic disease, one must be careful to distinguish between cause and effect. Although it is true that too much excitement will upset the stomach of a naturally highly-strung child, it is equally true that the child whose digestion has been thoroughly upset from a faulty or too liberal diet may show this upset by nervous manifestations. The whole problem of the nervous child is a most difficult one and should be approached by the physician with care and an open mind.

Nervous Exhaustion

The appearance of the so-called nervous-exhaustion child has been most aptly described by the late Leonard Guthrie and Hector Cameron.[1] The child, usually a boy, has a very characteristic stance, with all the signs of utter exhaustion, shoulders drooping, pelvis pushed forward, slight kyphosis of the back and hyper-extension at the knees (Fig. 60). All the muscles lack tone, those of the face are expressionless and appear "ironed-out," and the play of fine muscles about the

[1] H. C. Cameron, "The Nervous Child" (Oxford Medical Publications).

eyes and mouth has disappeared. As a rule the child is white-faced at all times, but when tired becomes quite pale and may look ghastly. Children to whom this description applies are subject in greater degree than the average child to nervous and metabolic disturbances, some types of which are described in detail below.

The management of the so-called "nervous-exhaustion children" is a difficult problem. They are often "only" children and are thrown too much into the company of their parents or elders, being constantly on their mental tip-toes as a result. The remedy lies in sending them as soon as possible, say at the age of 3½ or 4, to kindergarten, where they can associate with other children and be less in the limelight. To sing in the chorus rather than a continuous solo is what they really require. In this way they get mental and intellectual rest. They also require physical rest and should be kept off their legs during the morning. When possible they should lie down on their beds with the blinds drawn for at least one hour at midday. A certain proportion of these children are subject to acidosis attacks, for the treatment of which *see* p. 133.

Fig. 60.—Nervous-exhaustion child, showing position of general hypotonia. (*British Medical Journal.*)

Habit Spasm

This condition usually begins to appear at the age of about 3 years, and may continue for any length of time, even into adult life. It takes many forms, the most usual being blinking of the eyes, twitching of the corners of the mouth, shrugging the shoulders, sudden rotation of the head or skipping or hopping when walking.

Clinical picture.—The difficulty is to differentiate

between the movements of chorea and those of habit spasm. In chorea the more attention given to the child the worse the movements become, whereas in habit spasm the child, if he makes the attempt, is able to control the movements for the time being. In chorea the movements are never the same, the arms, legs, mouth, and, in fact, every muscle in the body, being involved, and the movements flit from one limb or part of the body to another. In habit spasm it is characteristic for the same movement to be repeated over and over again; that is, once shrugging has commenced, the same shrugging movement will be repeated hundreds of times, until a fresh tic sets in and the first one ceases. The child is usually thin, white-faced, over-active and mentally bright, and as a rule the offspring of "nervy" parents.

Etiology.—Nervousness or lack of self-control in childhood is as a rule a sign of general ill-health, and very often some slight organic lesion which has initiated a movement leaves the movement behind as a functional abnormality when the original lesion has disappeared. Thus a mild conjunctivitis leaves a habit of blinking, or a tight collar a habit of tilting the chin. A careful examination may in some cases, however, reveal fine tremors of the fingers, and there may even be slight exophthalmos with von Graefe's sign. Tachycardia and a tendency to flush may also occur. A proportion of children, then, in addition to habit spasm may be suffering from hyperthyroidism.

Treatment.—No good purpose can be served by drawing the child's attention to his disability. An attitude of studied unconcern should be adopted. Effort should be concentrated on improving the child's general health, and a careful examination should be made with this end in view. Bad teeth, septic tonsils, lack of fresh air or of sufficient rest should be remedied, and the diet should be reviewed to ascertain whether it is adequate and well-balanced. Undoubtedly a change of surroundings is more frequently successful than any other form of treatment. Tonics containing iron and

strychnine, or in winter months cod-liver oil and malt, are indicated. In cases where there is any indication of hyperthyroidism, a course of the French tincture of iodine, 5 minims in a little milk daily, appears to be most beneficial.

Anorexia (Lack of Appetite)

Lack of appetite in infancy and childhood may be due to a variety of causes, some organic and some functional.

From the doctor's standpoint the all-important cases are those due to *organic disease.*[1] As a rule there is some focus of sepsis, such as infected tonsils or bad teeth, which, by producing a chronic gastritis or gastric catarrh, probably brings about the loss of appetite. Certainly the removal of such a septic focus has most beneficial effects in the great majority of cases. Less common organic causes of anorexia are tuberculosis, rheumatism, including carditis and chorea, rickets associated with dentition, bronchitis and pyelitis. Lack of appetite may also mark the period preceding and following the onset of acute infectious diseases such as whooping-cough and measles.

About one-third of all cases of anorexia observed have no discoverable lesion, and in our present state of knowledge it must be concluded that the cause is *functional.* It is cases belonging to this class which present the most difficulty and, at the same time, the greatest interest to the physician. A certain number of such children require little more than an entire change in their surroundings. They are run down, and the particular district in which they live appears to be unsuitable for them. Others improve with the addition of vitamins to the diet; cod-liver oil and abundance of fresh fruit and vegetables seem to make all the difference. Again there are children who have developed bad habits of eating, or are negativistic, or whose parents expect them to have no appetite and

[1] W. M. Bartlett, "An Analysis of Anorexia," *Amer. Journ. Child. Dis.*, Vol. 35, 1928, p. 26.

who therefore live up to their reputation. These cases belong to the so-called *anorexia nervosa* group. Patients do best when removed from their home surroundings and faulty environment; then, benefiting by association with a tactful person or with healthy normal children, they themselves attain normal health. Thus one often finds that a puny, thin child who has had a poor appetite, and has failed to thrive, improves on entering a boarding-school, and within one term has gained weight and is eating satisfactorily. The improvement can only be attributed to the replacement of faulty home surroundings by healthy companionship and example at school.

There is, however, yet another group of cases of anorexia and these are the most difficult of all. It comprises the children who are constitutionally incapable of digesting or metabolizing normally large quantities of food. This disability is, as a rule, inherited, and the mother or father, or both, exhibit the same peculiarity. Such children are thin, under weight, and very often extremely active physically and mentally, and unless their activities can be curtailed they remain under weight and their progress is unsatisfactory. Their ability to digest and absorb more food improves as they get older, but even with a careful diet and perfect surroundings this incapacity for increased metabolism cannot be influenced. Any attempt to push food too far results in bouts of vomiting or diarrhœa.

Perhaps one of the most common causes of anorexia in children is *too exclusive a milk diet.* Large quantities of milk, cream, butter and eggs are being pressed on the child, who consequently refuses all other food. A diet high in fat invariably provokes a tendency to liverishness or acidosis, resulting in loss of appetite. For the correction of this condition *see* p. 134.

To summarize, in attempting to set a case of anorexia right, organic disease must first be excluded. This may be done by a careful examination of the throat and mouth and other organs, always including the

urine. Having excluded organic disease, the diet must be gone into, to see that it is well-balanced and that while it furnishes the necessary vitamins it does not contain too much milk, cream, and other fatty foods. A change of air or surroundings may be necessary, and iron-containing tonics should be prescribed for the anæmic, among the best being ferri carb. sacch., as much as will lie on a sixpence three times daily; Parrish's Chemical Food, half a teaspoonful three times daily; liver, once or twice weekly; raw or underdone scraped steak, a dessertspoonful two or three times weekly. Where it is tolerated, cod-liver oil in the form of one of the preparations, such as Bynol, Scott's Emulsion or Radiomalt, should be given, a small teaspoonful three times daily after meals; or Ostelin or Radiostol may be employed, 2 to 3 drops three times daily after meals.

Disturbed Sleep and Night Terrors

Among the causes of disturbed sleep in the **young infant** *nasal obstruction*, due to a cold or catarrh of the nose, is the most common. *Overfeeding*, especially when the child is not held up afterwards to break or bring up his wind, tends to cause colic and indigestion, and with each bout of colic during the night the child tends to cry out. *Underfeeding*, with consequent hunger-pains, will likewise disturb the child continually. Some children *when cutting teeth* appear to have neuralgia, and this apparently causes them to cry out at intervals. Some require bed-socks or hot-water bottles, otherwise they wake early and cannot be induced to go to sleep again.

In the **older child**, who fails to go to sleep until very late, the cause is probably *insufficiency of fresh air* during the daytime, and the child gets into the habit of sleeping late in the morning to make up the deficiency of sleep. This should be rigidly discouraged. Night terrors are most often due to *adenoids*. A common history is that, following on a cold, the child woke each evening once or twice, screaming out. This

gradually passed off, to recur some months later with another acute nasopharyngeal infection. The treatment is obviously removal of the adenoids. *Digestive disturbances* may also give rise to night terrors and disturbed sleep in the older child, the trouble being caused as a rule by too large a supper on going to bed. In cases of this kind tea-supper at 5 p.m. should be the last meal. A rare but interesting type of case is that in which the child wakes with what appears to be a night terror, but is probably a *hypoglycæmic attack*. The administration of two or three teaspoonfuls of glucose at bedtime, taking care to clean the teeth afterwards, prevents these attacks. Some children are wakened at intervals during the night by *passing their urine*, and the cold, wet bed tends to keep them from resuming their sleep. With a meatal ulcer, causing pain on micturition, this is very easily understood. Another possible cause of night terrors, which must always be borne in mind, is *nocturnal epilepsy*.

Nervous Vomiting

In making the diagnosis of functional or nervous vomiting the greatest care is necessary. The *organic causes* of vomiting such as cerebral lesions, including cerebral tumour and meningitis, must first be excluded. *Obstruction of the œsophagus and stomach* can readily be ascertained by means of X-rays. Among the *toxic causes* are acetonæmia and the action of certain drugs such as ipecacuanha. Among the *digestive causes*, one must consider gastritis and indigestion, the latter being due to an unsuitable diet. *Mechanical vomiting* includes vomiting induced by sucking the fingers, or by excessive coughing as in whooping-cough, and the retching, especially seen in the morning, due to infected adenoids. When all these have been carefully excluded, there remains a small group of cases in which the child, usually from the age of 6 months onwards, apparently can and does vomit at will. The older child, it is to be noted, never vomits over himself, and only vomits in the presence of some adult. In nearly all these cases there

has been originally some organic cause of the vomiting, such as tonsillitis or gastritis, and, too much notice having been taken of this, the child quickly learns that he can attract attention by the trick referred to and accordingly continues to vomit.

In treating an older child the mother and nurse must be taken fully into the confidence of the physician, and it is often best to give a bismuth meal and have the patient screened in front of them, so that they can see the bismuth passing rapidly through the stomach. Thus reassured, they can tackle the vomiting problem with a firm hand and studiously ignore it.

Masturbation

This practice may be indulged in at any age, and is in fact relatively common among female infants from the sixth or eighth month, the act being performed by rocking back and forth, rubbing the thighs together, crossing the legs and so on. In males it is uncommon before the age of 3 to 6 years.

The fear in the mind of the mother or nurse is that this act shows a tendency to excessive sexual precocity or to some vicious tendency. This fear, however, is quite unfounded, and the whole thing should be looked upon as a reflex act of a pleasurable nature, on a par with thumb-sucking. Sex does not enter into the question at all until the child is much older.

In the treatment and management of such a case a careful examination should first be made to exclude any possibility of local irritation. In the female this may be caused by want of cleanliness or adhesions between the labia. In the male an overtight prepuce or slight inflammation may be present. Sitz baths for the female child with a little Condy's Fluid to colour the water, and in the case of the male child circumcision, will probably set the matter right. Where no sign of local trouble is to be found, the child should be carefully watched at his resting period, at bedtime or on waking, as these are the times when the practice is most common. The child's mind should be kept employed or diverted

from the generative parts at such times by some artifice such as giving him a toy to hold. The author has known cases cured by giving the child a spool and a rubber band, so that he employs his mind winding the band round the spool and unwinding it again until he falls asleep naturally.

It is essential to make it quite clear to the mother that sexual precocity is not involved, and that no permanent harm is caused by this act. There is a widespread belief among the public that masturbation leads to mental defect, but this is, of course, quite unfounded. Masturbation is extremely prevalent, and at some time or other has affected almost every individual. It occupies a short or a long phase, depending on the type of child or the fuss made about it by the parents. Where those in charge show great anxiety and remonstrate with the child, it is probably persisted in for a considerable period. On the other hand, where very little notice is taken and the child is kept healthily employed, he will quickly pass through this phase.

Thumb-sucking

The evils of thumb-sucking have been much overrated, and at worst it can only be regarded as a rather dirty habit. In the first few weeks of life the child's thumb or fingers may have been placed in its mouth to ensure that it remains quiet, especially during the night, and the habit is quickly established. In little children thumb-sucking may be practised throughout the day and night, but in older children it is done as a rule only when they are tired or going to sleep. Results such as protruding teeth, adenoids, nasal obstruction and a high-arched palate are all attributed to thumb-sucking, but there seems very little evidence in favour of this; in fact many thumb-suckers appear normal in every way, and many children with adenoids have never sucked either their thumbs or a dummy throughout life. The chief objection is the possibility that the child may infect himself with thrush or some pathogenic organism. The best treatment is to substitute some

other habit, or to distract the child's attention from the one under discussion. In infants it is sufficient to put cotton gloves on the hands or to bandage them, as the child quickly forgets. For older children a variety of apparatus has been devised. The child can often be "shamed" out of the habit, or the thumb can be painted with some disagreeable substance such as arnica each night. The author's feeling is that this habit is of little practical consequence and that it is wrong to take too much notice of it.

Stammering

Stuttering and stammering are among the most serious functional disabilities of childhood, and should be regarded by parents with the greatest care. In the nervous type of child in which it appears, it is a sign that he is run down and requires a change or a holiday. On no account should his disability be pointed out to him or stressed in any way; rather, as in other forms of habit spasm, he should be given some occupation which will tend to correct his fault. If old enough he should be made to read aloud slowly, or if too young to read he must sing or recite pieces of poetry over and over. In this way he rapidly regains confidence in his ability to speak without stammering. It is only in the older child, of say 7 or 8 years of age, that a voluntary method of treatment should be resorted to. Then the services of an elocutionist and gymnast, by educating the child in correct breathing and phonating, will help to overcome the habit. The greatest chance of success, however, in the early stages, up to the age of 4 or 5 years, lies in treating the matter with apparent unconcern and relying upon re-education on the lines described, with special attention to the child's general health.

CHAPTER XIV

DISEASES OF THE BONES AND JOINTS[1]

Congenital Dislocation of the Hip

Congenital dislocation of the hip is a very rare condition, and its etiology is unknown. Though present at birth, it is usually unnoticed before the child commences to walk at the age of about a year or 14 months. It is then seen that the child walks with a limp and that one hip is higher than the other. If laid flat on the back, abduction and external rotation of the hip are limited on the affected side. If when lying flat on the back the knees are flexed, leaving the feet on the ground, the knee on the affected side is lower than on the unaffected side. X-rays readily confirm the diagnosis. Occasionally the condition is bilateral, the child walking with a waddling gait and exhibiting a marked lordosis. The treatment is purely surgical and in most cases proves very satisfactory.

Flat-foot and Knock-knee

A very common defect, especially among the well-to-do, is knock-knee and flat-foot, the result of mild rickets in infancy. The child is over weight, fat and flabby, and by the age of two is seen to be both knock-kneed and flat-footed. Prophylactic treatment should be adopted early, and no child should be allowed to become grossly overweight. Strenuous *antirachitic* measures should be taken, including sunlight treatment, either artificial or natural, cod-liver oil being given and the diet as a whole curtailed. Massage is also of value. If the condition is established when the child comes under

[1] John Fraser, "Surgery of Childhood" (Edward Arnold & Co.), p. 215.

observation, the following steps should be taken: The *diet* should be reduced if the child is over weight, especially as regards milk and starchy food. A little *cod-liver oil* emulsion, artificial or natural *sun-baths*, and *massage* if possible, should be instituted. To correct the tendency towards *flat-foot* the heels of the shoes should be wedged, so that the weight of the child is thrown on to the outer part of the foot. This exerts a corrective influence on the knock-knee at the same time. In altering the shoes, the heel should be removed and a wedge $\frac{3}{16}$ inch thick, tapering away to nothing, inserted, the thick end of the wedge on the inner side of the heel. The heel of the shoe is then replaced. Shoes or boots which fit snugly around the ankle and heel are necessary, and some such shoe as the "Start-Rite," or any other make that is designed to support the instep, is desirable.

Night splints, the angle of which is gradually altered with a key and ratchet, in addition to the wedged shoes, are of great value in severe cases of *knock-knee*.

Faulty Posture

The so-called nervous-exhaustion stance, referred to in the preceding chapter, is exceedingly common. Here the shoulders droop forward and the child stands with the pelvis pushed out. The chest is flat and the back rounded. There is a tendency to hyper-extension at all the joints and the muscles are hypotonic. These children are of the highly-strung type and are easily tired. They are able to maintain an erect and becoming posture for a short time only, and quickly relapse into this exhausted or fatigued stance. Such children are said to be round-shouldered.

The treatment should aim at the general improvement of the child's health, ensuring frequent short rests, and plenty of sleep during the night hours. For dietetic measures *see* the treatment of fat dyspepsia, p. 129. Breathing exercises, tonics, changes to the seaside, artificial or natural sunshine and other steps to improve and tone up the child's whole musculature

should be undertaken rather than too localized a treatment of the particular group of muscles affected.

Spinal Curvature

This defect may be due to organic disease of the spine, such as spinal caries (tuberculous) (Fig. 61), or a congenital malformation of the vertebræ (Figs. 62, 63), such as absence of a vertebra or the occurrence of wedged vertebræ. On the other hand it may be due to some affection of the spinal muscles, such as that following on poliomyelitis. Rickets may produce a kyphosis which easily undoes itself if the child is placed on his face or held up under the arms.

Fig. 61.—Spinal caries.

Static curves (scoliosis or kyphosis) are exceedingly common, though the etiology remains obscure. Some of them may be due to a short leg, or may date back to some slight injury of the back-muscles or early rachitic deformity. Chest infections or torticollis may also produce an abnormal stance.

Treatment.—Once organic disease has been excluded, the treatment of which must be dictated by an

Fig. 62.— Spinal curvature in a girl aged 9 years with 14 ribs on one side and 12 on the other.

orthopædic surgeon, there remains a group for which no organic cause can be found. Swedish drill, breathing exercises, swimming and hanging from parallel bars, all tend to improve such cases. It may be necessary in the most severe cases, where there is actual secondary deformity of the vertebræ, for the child to sleep at night in a plaster bed.

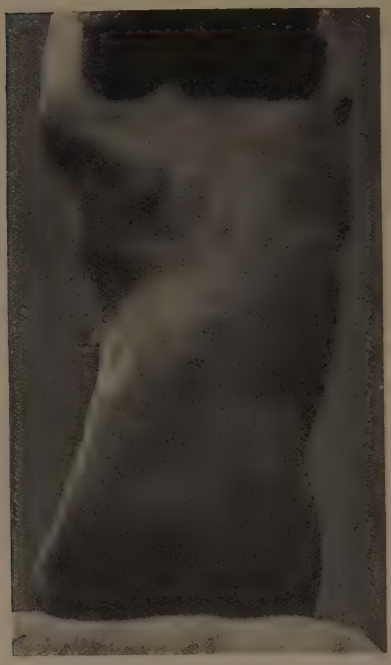

Fig. 63.—Same case as Fig. 62.

Achondroplasia

This is a rare hereditary disease, presenting a characteristic clinical picture, which should be recognizable from birth onward. The most characteristic features are the shortness of the arms and legs compared with the trunk and the large square head (Fig. 64). The hands are fat, and there is a tendency for the little and ring fingers to cling together, leaving a space. The middle and index fingers are also approximated and the thumb stands apart—the so-called "trident hand." Mentally, achondroplasic children are normal or even unusually bright. The progress in adult life is normal in every respect and both males and females are normal sexually.

The essence of the pathology is, as the name suggests, that there is an interference with endochondral

ossification, especially at the extremities of the long bones.

Treatment.—There is no rational treatment known.

Fig. 64.—Case of achondroplasia aged 10, height 37 inches.

Fragilitas Ossium (Brittle Bones and Blue Sclerotics)

This is an extremely rare familial complaint. In infancy it is very difficult or impossible to diagnose, but

as the child gets older and appears to be developing normally, there is a tendency for fractures to occur repeatedly. It is also noticed that the sclerotics are extremely blue. After the age of 4 or 5 years the head is observed to be getting larger in the temporal regions, so that there is a marked bulging above the ears. Any other deformities which may occur are due to malposition after fractures. Some children fracture so infrequently that they are able to lead almost normal lives, but in others the tendency to brittle bones is so pronounced that they are almost complete invalids. Liability to fracture appears to grow less as years go by, the children tending to grow out of the disability in adult life.

Nothing is known as to the *etiology* of the disease. The *pathology* shows a deficiency of the fibrous trabeculæ binding the osteoid tissue together. Various forms of treatment have been advocated, including the giving of calcium and other antirachitic measures. An examination of the blood, however, shows that the blood-calcium and blood-phosphorus are normal, and antirachitic measures are not specially indicated. The child should lead a careful life, free from rough exercises and games. Great care should be taken with each fracture, that shortening of the limbs and malposition are not incurred. No rational treatment has yet been described for this disease.

Osteogenesis Imperfecta (Fig. 65)

This rare complaint is sometimes spoken of as "congenital rickets." Actually the child is born with imperfect ossification of many of the bones, especially those of the skull. The head feels like a bag of jelly, and an X-ray of the bones shows marked osteoporosis (Plate 15). There are usually many fractures of the ribs, dating from birth, and fractures of the arms and legs occur with even the gentlest handling, such as dressing and undressing the child. In one case recently seen by the author both femora were broken when the

Plate 15.—Radiogram in a case of osteogenesis imperfecta (same case as Fig. 65).

child was gently placed on his chamber in an effort to teach him clean habits.

The etiology of this condition is not understood. There is apparently a very serious deficiency in the bone calcification, but the blood-calcium and blood-phosphorus would appear to be quite normal. Internal secretions have been blamed for the condition, and thyroid, pituitary and other glands given. The author has seen no good results, however, from either anti-rachitic or gland treatment. The child is best nursed on a large pillow and handled as little as possible. Such infants are very liable to die from intercurrent

Fig. 65.—Case of osteogenesis imperfecta, aged 18 months.

complaints, such as pneumonia or influenza. The outlook as to life is extremely bad, very few surviving the second or third year.

Osteomyelitis

This disease is rare, but on account of the extreme importance of early diagnosis it is worthy of mention. The organism in the vast majority of cases is the staphylococcus aureus, but occasionally the pneumococcus or the streptococcus is responsible. The route of infection is almost certainly via the bloodstream. There is often a history of some superficial infection, such as boils or abrasions of the skin, followed by bone infection. The metaphysis of the bone is the original site in most cases. The disease is most commonly found between 3 and 10 years, and it reaches its maximum at the sixth year.

Fig. 66.—Femur from a case of osteomyelitis.
A, path taken by infection into knee-joint.
B, primary focus of infection.
C, path taken through necrosed bone to popliteal space,

The bones most frequently affected are the tibia, the femur and the humerus, in that order (Figs. 66, 67).

Clinical picture.—Following some infection, such as boils or tonsillitis, after an interval varying in length, the child complains of pain in the affected bone. The onset is sudden and severe, accompanied by fever, and is confined to one end of the bone. At once it is noticed that the child is gravely ill, showing a flushed face and a dirty tongue, a rapid pulse and, in fact, all the clinical symptoms of acute toxæmia or poisoning. There may even be rigors. On examination of the affected part, at first there is very little to be

seen, but gradually a little swelling or œdema appears and finally there is a distinct reddening in addition, suggesting a deep abscess. There is much pain on handling or moving the limb.

Differential diagnosis.— In making a diagnosis such conditions as erysipelas, acute rheumatism and cellulitis of the deep tissues, or inflammation of some gland (adenitis), must be excluded. With none of these is there so much general toxæmia and upset, however. It is a golden rule when in doubt in such a case to operate.

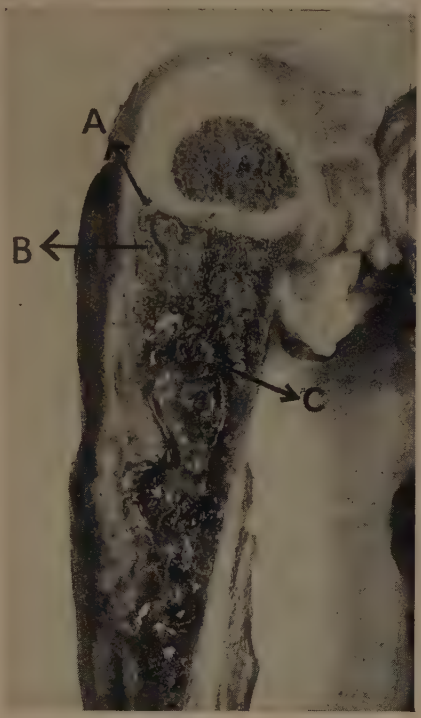

Fig. 67.—Osteomyelitis. (*See* **Fig. 66.**)

Treatment.—The treatment is purely surgical and should on no account be delayed. Transfusions are found most helpful in such cases.

CHAPTER XV

DISEASES OF THE SKIN

Napkin Rashes

Rashes in the napkin area may be either a local manifestation of some general disease or merely an indication of neglect or mismanagement of an otherwise normal infant. They may, however, be due to the action of chemical irritants, most frequently acid stools, or alkalis liberated from the urine.

CONGENITAL SYPHILIS

This is a rare condition. Occasionally at birth, but much more commonly between the fourth and eighth week after birth, the buttocks are noticed to be red and glazed, and to assume a copper-coloured tint. Round the anus condylomata appear, with moisture on the opposing surfaces of the nates. With treatment the rash disappears quickly, but it does so spontaneously in any event after several weeks. It should be remembered that the rash is not confined to the buttocks in congenital syphilis, but is present elsewhere on the body, the face, the palms of the hands and the soles of the feet. This is a very important diagnostic point.

Treatment.—The treatment is that for congenital syphilis (*see* p. 484).

EXCORIATION OF THE BUTTOCKS (DERMATITIS)

If the buttocks are allowed to remain wet for any length of time the skin is inclined to become sodden, and is then open both to infections and to the destructive effects of weak acids and alkalis. One of the most

frequent causes of ulceration of the buttocks is simple neglect. In such cases the infant is not changed for many hours at a time, and the buttocks become raw as a result. There are, however, two definite forms of this dermatitis—(*a*) acid dermatitis, (*b*) ammonia dermatitis.

(a) Acid dermatitis.—*Excessive carbohydrate* (sugar or starch) in the diet is liable with undue fermentation, especially in warm weather, to break down into diacetic acid. The stools are frothy and very acid to litmus, burning and excoriating both the bowel-wall and the buttocks with which they come in contact. If these acid motions continue it is almost impossible to prevent them from burning the child, unless the napkin is changed immediately the motion is passed. *An excess of fats*, with a fat dyspepsia, will produce a picture exactly similar. At first the stools are pale, formed and crumbly, later they become loose and light-coloured, and finally curdled, acrid in odour and strongly acid, with much free fatty acid (butyric acid).

Treatment.—If the diet contains too great a quantity of carbohydrate or fat, this must be temporarily reduced. It should not be forgotten that infections may be the causal factor in lowering the child's tolerance of fat or sugar for the time being.

Local treatment of the buttocks consists in frequently changing the child, taking care, in cleaning the buttocks, not to use a rough towelling napkin which might injure any fresh skin that is beginning to form. Pads of cotton-wool, changed frequently and left inside the ordinary napkin, will be found most useful. A mixture of zinc ointment and castor oil, applied to the buttocks during the night 12 hours, is a suitable preparation, as it prevents the urine or the liquid stools from adhering to the skin, which does not therefore become sodden. During the day 12 hours an attempt should be made to dry up the whole area of the buttocks by applying freely zinc oxide and starch powder or some other good dusting-powder such as Johnson and Johnson's, or Dermatol.

(b) Ammonia dermatitis.—Some infants on being

wakened in the morning smell strongly of ammonia, and on examination it will be found that the buttocks are reddened. Later, if neglected, these parts actually become burnt. There are several causes of ammonia dermatitis. If *too large a quantity of creamy milk* is given in the day, in an attempt to counteract the acidosis, much ammonia is excreted by the body, and when this comes into contact with the soap of the napkins or sheets free ammonia is liberated. In such a case the milk should first be skimmed, in this way reducing the total quantity of fat given in the day. Napkins should be rinsed thoroughly to get rid of the soap. Another cause of ammonia dermatitis is the *micrococcus urease* or other urea-splitting organism commonly found in the stools. The action of these organisms on the urine-sodden napkin, it is thought, liberates ammonia which burns the buttocks. Dipping the napkins in a 1-in-4,000 solution of perchloride of mercury, which acts as a mild antiseptic, is sufficient to destroy the organisms. Possibly the alkali which liberates the ammonia from the urine is supplied by the alkaline stool itself, and therefore the proper prophylactic treatment lies in training the child to pass a motion at the proper time into a chamber, avoiding defæcation during the night hours.

Chilblains (Erythema Pernio)

Chilblains manifest themselves by swelling, redness and tenderness of some or all of the extremities. There is little doubt that the chief etiological factor is sudden change of the temperature of a limb from cold to hot. Children with very cold hands and feet who plunge them in warm water, take very hot baths or are allowed to warm these extremities before the fire often develop chilblains. Those subject to chilblains usually have very poor circulation and a low blood-calcium.

Treatment.—When the hands or feet are particularly cold, especially if the child is predisposed to chilblains, care should be taken to warm them gradually. They should be guarded against cold with warm gloves

and stockings, and this is an important prophylactic measure. Calcium lactate, 3 to 5 grains daily, or calcium chloride, 3 grains three times daily, may be given as medicinal treatment. A good preparation is Kalzana, a half-tablet 3 times a day being a suitable dose. Parathyroid, $\frac{1}{10}$ grain to $\frac{1}{4}$ grain three times daily, is reputed to be valuable. Tonics such as cod-liver oil, artificial sunshine and a change to the seaside should complete the cure.

Parasitic Eruptions

SCABIES

The infection is caused by the female acarus. The eruption is seen on the hands and feet, most commonly between the fingers and toes, and also in the folds behind the knees, in the groins, beneath the arms and about the perineum. Small blebs form at the site of the burrow. There is frequent itching, and as a result the bleb is torn open, leaving a tiny abrasion. The irritation is greatest during the night or when the child is extremely warm. Infections from the skin are liable to be superimposed on these burrows, masking the true state of affairs. Scabies is seldom seen on the face. The disease resembling it most closely is lichen urticatus or urticaria papulosa. This latter condition is seldom seen between the fingers and toes, however, and is often found scattered over the trunk, especially in the loin region.

Treatment.—In tiny infants washing gently with soap and water and applying friars' balsam to all the spots rapidly removes the infection. Clothing and bedding must be sterilized and sources of infection such as the mother or nurse must be eliminated. In older children the treatment should begin with a warm bath, and a good scrubbing with soap and water. The application of sulphur ointment (B.P.), either full strength in the case of an older child, or diluted with vaseline, equal parts, in younger children, should be used. This must be applied all over the body, and

reapplied at intervals for three days. During this period the same underclothing or sleeping suit should be used; this becomes impregnated with the ointment and rubs over the skin surface, working the ointment actually into the burrows. By the third day another warm bath should be given, and the bedding and clothing should be sterilized, and then, as a rule, a cure is soon effected. It may be necessary, however, to repeat this treatment a fortnight later.

Where the picture is much complicated by secondary infection a cure may be delayed, and treatment for dermatitis or an eczematous or impetiginous condition may have to be undertaken first or simultaneously.

PEDICULOSIS CAPITIS

This condition was at one time very common, but has recently been much less so, owing to the mode of keeping girls' hair clipped short. "Nits" can be seen fastened to the hairs, and are easily distinguished from scurf or dandruff, which is not fastened to the hair. Infestation leads to scratching of the head, and erythematous rashes may be present over the neck and forehead, or there may actually be ulceration of the scalp. Usually, however, pediculosis capitis is accompanied by impetigo of the scalp with large crusted areas. The glands of the scalp and neck draining the infected scalp area are often enlarged.

Treatment.—Soap and water, with a fine tooth comb to remove the nits, are all that is required in slight infections, but where the infection is great, oil of sassafras should be sprinkled over the hair after careful washing of the head, and a towel wound round it. In 12 hours, with a further combing and washing, the nits should come away and the condition right itself. Washing the hair and scalp in petrol, benzine or paraffin is very efficacious, but dangerous, and requires to be carried out with the greatest care. It is doubtful whether the risk is warranted. For the treatment of the accompanying impetigo *see* p. 378.

RINGWORM (TINEA)

Tinea corporis (ringworm of the body).—This is often communicated from one child to another, or by the agency of a cat or dog. It does not spread to the scalp or nails. Small areas with a raised edge, aptly described by the name *tinea circinata*, form on the forehead, chest, back, arms, legs, etc., and rapidly spread. The diagnosis is best made by the microscopic examination of scales of epidermis for the mycelium.

Treatment.—The application of any antiseptic ointment, especially tincture of iodine, dilute ammoniated mercury or Whitfield's ointment (salicylic acid 10 grains, benzoic acid 10 grains, coco-nut oil ½ drachm, and vaseline ½ oz.), forms the treatment for this complaint.

Tinea capitis (ringworm of the scalp).—This is commonly seen in children, and is transferred from child to child by hair-brushes, combs or caps. Small or large areas can be detected, in which most of the hairs are broken short. Gradually the whole scalp becomes involved, and the denuded areas may be scaly or crusted. The clinical diagnosis can be confirmed by the extraction of the hair-stumps and a microscopical examination of these.

Treatment.—1. The hair should be cut short and the infected areas must be inflamed by means of local applications. Freezing with ethyl chloride is most efficient, and this treatment should be repeated at intervals until the whole area is thoroughly red and inclined to be indurated. Ung. hydrarg. nitratis (B.P.) is also useful for this purpose.

2. X-ray treatment, causing epilation and the rapid cure of the condition, has been extensively used for some years. In the hands of a skilful worker this method is both safe and most efficient. The dangers of alopecia can be minimized.

3. The removal of the hair by internal administration of thallium acetate has recently been described. The dose is 4 mgm. of thallium acetate per pound of body-

weight. In a period of from one to two weeks the hair falls out. There is no tendency for alopecia to result, and in this way it is a great advance upon X-rays. The disadvantage, however, is that it is easy to overdose a patient, giving rise to toxic symptoms, with vomiting, headache and albuminuria. With further experience, however, this method would appear to be the most suitable for the treatment of children.

Tinea unguium (ringworm of the nails).—In this manifestation of ringworm the nails are wrinkled, pitted and discoloured. Removal of the nails is the surest way of curing the condition, otherwise it may persist for months or years.

Impetigo Contagiosa

This is due to a streptococcal infection of the skin, and, as its name suggests, is extremely contagious. It appears especially about the face, and its outstanding features are the formation of crusts, and a tendency to spread to the hands, the knees, behind the ears and into the hair. The crusts appear as if "stuck on." Often, as has been previously stated, impetigo contagiosa is associated with pediculosis capitis, and the occipital glands become enlarged and inflamed and may actually break down.

Treatment.—Crusts must not be allowed to form if treatment is to be successful. If they have formed on the face, a starch poultice in the shape of a mask must be applied until the crusts have been removed, then half-strength ung. hydrarg. ammon. dil. and vaseline should be applied continually. If the lesions are amid the hair, it is best to clip or shave the hair away from the infected parts, then foment. Ultra-violet light is said to shorten the course of the disease.

Bullous impetigo is a staphylococcal infection of the skin, sometimes seen as a *whitlow* about the finger, or as *pemphigus neonatorum* in the new-born. For a description of the latter *see* p. 39.

Furunculosis (Boils)

This disease is due to the staphylococcus pyogenes aureus, the infection being in or round a hair follicle. Any condition causing a hyperglycæmia tends to produce them. Overfeeding, especially on fats or sugars, may be a contributing factor. The lesion may take the form of very fine pustules or even of indurated boils. Common sites are the buttocks, the region of the perineum, under the arms, the back of the neck or the scalp. Boils are especially common in children who wet the bed and in school-children who get an insufficient number of baths.

Treatment.—The area must be fomented, care being taken that the pus is not allowed to drain on the surrounding parts. At intervals the surrounding skin should be wiped over with a weak antiseptic lotion. If the boils are present in the perineum or on the buttocks, the underclothes should be lined with silk at those parts to prevent friction. Injections of stock or autogenous staphylococcal vaccine are extremely valuable; three should be given, one every third or fourth day. Treatment for enuresis should be instituted.

Seborrhœa (Scurf of the Scalp)

In little infants, especially of the hospital class, scurf tends to form down the centre of the scalp and to collect, usually over the anterior fontanelle.

Treatment.—The scurf is best removed with soap and water or soaked off with olive oil. A fine tooth comb should then be used to comb away the scales.

Warts (Verrucæ)

These are infectious and have lately been shown to be due to a filter-passing virus. They are most commonly found on the hands, face and scalp.

Treatment.—Glacial acetic acid with 1-per-cent. perchloride of mercury, applied by means of a match-stick to the wart, is most efficient. Freezing with carbon dioxide snow is effective, as is the use of X-rays.

Lupus Vulgaris

Lupus is comparatively rare in children. It usually appears on the cheeks and nose, and is probably due to direct infection from phthisical parents kissing their children. It may, however, appear in any part of the body. At first there is a small tuberculous nodule, which gives rise to an indurated patch. This may remain for some months.

Treatment.—Adamson[1] recommends that treatment should consist of either (1) an attempt to remove the disease at one operation under a general anæsthetic by excision or scraping, or (2) a more gradual removal of the lesion by selective caustics or light therapy, combined with general treatment. Where small patches only exist, excision should be attempted. The caustics he suggests are strong pyrogallic acid ointment, 40 per cent. in vaseline, or liq. acid nitrate of mercury. Ultra-violet light may be of the greatest use, and Finsen light, although expensive, is ideal.

Herpes Zoster (Shingles)

This is due to an infection, by an ultra-microscopic virus, of the posterior root ganglia. It is interesting to note the connexion between chicken-pox and herpes. Very often after an adult case of herpes a case of chicken-pox follows in the household, or vice versa.

Symptoms.—The eruption is formed of vesicles and is distributed along the sensory fibres of a posterior nerve root. The chest, abdomen, thighs, neck or forehead may be involved. An erythematous rash appears first which becomes vesicular, the vesicles containing clear fluid. Later these dry up and fall off, this stage being accompanied by a slight fever and some degree of malaise, and occasionally neuralgic pains. Scarring does not occur if the scabs are left alone. Herpes of the fifth nerve may be accompanied by corneal ulcer and iritis, in which event great damage to the eye occasionally results.

[1] H. G. Adamson, in Garrod, Batten, Thursfield and Paterson's "Diseases of Children" (1929), p. 1014.

Pathology.—Lumbar puncture shows lymphocytosis of the cerebro-spinal fluid, and on section the posterior root ganglia show inflammation and hæmorrhage.

Treatment.—Friction and scratching of the lesion should be prevented, and it should be kept dry with zinc-and-starch powder. Painting with collodion is also useful. A little aspirin may be given at bedtime if the child is restless.

Herpes Febrilis (Herpes Recurrens)

This lesion, usually appearing about the mouth or nose, is an accompaniment of the common cold. It shows itself by a crop of small vesicles. Other sites are the cheeks, the ear, the neck or the fingers. It may accompany pneumonia, cerebro-spinal meningitis, tonsillitis or intestinal upsets. The vesicles dry up in a few days and fall off. It tends to recur at the same site.

Etiology.—Herpes has been shown to be due to an ultra-microscopic virus.

Treatment.—Similar to that described for herpes zoster.

Erythemata

Toxic erythemata may resemble the rash of measles or that of scarlatina. They differ, however, in the fact that there is no fever, that parts of the body are quite free of the rash, and that neither coryza nor sore throat is present. There is usually a history of food-poisoning.

Erythema nodosum.—For many years this was thought to be of rheumatic origin, but recently two other views have been expounded: one that it is a specific disease due to a specific organism, and the other, that it is a skin manifestation of a tuberculous bacillæmia.

Symptoms.—Red, tender, oval swellings appear on the front of the shins. There may be several on each shin, and occasionally they are also found on the

forearms. The onset is marked by malaise, joint-pains, shivering and sore throat, with a rise of temperature. The nodes gradually disappear, fresh ones appearing for some few weeks.

Course of the disease.—There is often a second bout after the first has subsided.

Prognosis and treatment.—The child should be kept in bed, with the legs protected from the bed-clothes. Aspirin should be given by the mouth and lead lotion applied locally. A large number of cases have now been reported where some tuberculous lesion has developed afterwards, and most of the subjects react strongly to the tuberculin tests, especially the Mantoux test. This being the case, measures should be instituted after the infection to build the child up as much as possible.

Urticaria (Lichen Urticatus, Urticaria Papulosa, Heat-Spots, Nettle-rash, Hives) [1]

Etiology.—The cause of this affection is not entirely understood, but it appears to be bound up with the following factors:

1. *General overfeeding.*—The spots, although they may appear throughout the year, are very much more common in the summer months. It is in the warm weather that children are most liable to be overfed. The great majority of those affected by urticaria are over weight or inclined to be rather plump, though it does occasionally occur in the thin child, and it should be taken as a sign that the digestion is being taxed to the limit of its powers.

2. The overfeeding may be on "*heating*" *foods*, such as porridge, groats, yolk of eggs, cream or much butter.

3. Strawberries, cherries, plums, raspberries, gooseberries, bitter oranges or lemons may all *produce acidity* and thus bring about the spots.

[1] Donald Paterson, "Heat Spots in Children," *Nursing Mirror*, Aug. 22, 1925.

4. Shell-fish, rabbit, ducks' eggs, meat-juices may also produce these lesions in some *susceptible children.*

Clinical picture.—The spots may be typical of nettle-rash, with a slightly raised red area which irritates. On the other hand, they may resemble the bite of an insect. Common sites are the backs of the hands, the forearms, legs, loins and face, but they may appear on almost any part of the body except the scalp. The common type appears as small, shotty, hard, white blisters, which when scratched contain clear fluid and give place to a small raised inflamed or red area. These may be mistaken for early chicken-pox. Both types are known as "heat-spots," "nettle-rash," "hives," or "teething spots." If the tops are torn off the spots may become infected.

Treatment.—A careful review of the child's diet should be made, in order to ensure that he is not being overfed. The best course is to adopt the principle that the less often he is offered food the less likely he is to be overfed. Three meals a day only should be given after the age of one year. If the weather is warm, heating foods should be stopped, and groats, cream, butter, eggs and milk should be limited. Cod-liver oil and malt is often the offender. Raw fruit, especially very acid raw fruit, should be stopped. Tangerines are a good substitute for oranges, as they are less acid. Shell-fish, raw meat-juice, ducks' eggs, etc., must be avoided. One heaped teaspoonful of bicarbonate of soda should be given spread over the day, a pinch with every drink. The bowels should be kept well open with grey powder at bedtime and milk of magnesia the following morning. The spots should be dabbed with calamine lotion to allay the irritation.

Drug Rashes

Bromide rash.—Bromide is often supplied to a child in the form of "gripe water" or soothing powders, or the mother may be taking the drug for some purpose and, excreting it in her milk, transmits it to the baby. The rash appears as vesicles on the face, body and

limbs, and later these become hard, raised crusts, varying from the size of a pea to that of an almond. When they fall off, however, they leave no scar, and the lesions disappear rapidly with the cessation of the drug.

Belladonna rash.—This is scarlatiniform in character, and is accompanied by dilated pupils, dryness of the mouth, sometimes vomiting, rapid pulse and excitement.

Serum rash (serum sickness).—A rash may follow after injections of antidiphtheritic, antistreptococcal, antitetanic or other sera. As a rule it appears about a fortnight after the injections, but it may come earlier or later. It is often accompanied by an urticarial rash, which may be blotchy and erythematous, resembling measles. There is tenderness in the joints, a temperature, malaise and occasionally actual vomiting, these symptoms persisting perhaps for two or three days or longer. The joint-pains, with the subsequent rise of temperature, have been known to last as long as six weeks. Calcium lactate, 5 gr., or small repeated doses of chloral should be tried in such cases.

Enema rash.—This is liable to appear after the administration of an enema. The cause of the rash is not clearly understood. It is bright red, and appears on the knees, elbows, buttocks, face and chin. It may be a blotchy erythema or actually scarlatiniform. Most frequently it occurs within 12 hours of the administration of the enema, but may appear sooner.

Eczema

The term eczema is gradually becoming more and more restricted. At one time it included a large number of skin infections such as impetigo.

Etiology.—There are various theories as to the causation of eczema, none of which is definitely proven.

1. The *anaphylaxis theory.* It has been suggested that eczema, like urticaria, is due to an anaphylaxis to some food protein. Such foods as eggs, cereals, etc., are specially mentioned. In the breast-fed infant, it is suggested, the anaphylaxis is passed through the

mother's milk and is due to some foreign protein she is herself ingesting.

2. *Local inflammation* of the skin by various organisms due to skin irritation. It is suggested by many pure dermatologists that metabolic disturbances play no part in this condition; in other words, the type of food ingested and the incidence of teething, etc., are mere coincidences in accentuating the skin condition. This is not the general opinion among pædiatricians. The theory that irritation of the skin with infection by some low-grade type of organism is the primary and sole cause hardly meets the case, as metabolic disturbances and the particular type of child would appear to be equally important.

3. *Constitutional diathesis* (exudative diathesis). It is believed in some quarters, especially on the Continent, that there is a particular type of child who is prone to catarrhs of all the mucous membranes and who possesses a low immunity to infections. This theory presupposes (*a*) appropriate soil, that is, the exudative diathesis or catarrhal child, and (*b*) local irritation of the skin with abrasions. To the author this seems nearer the truth than any other explanation so far advanced. Children of this type have an extremely low fat-tolerance, and if this is exceeded the catarrhs are accentuated and immunity further lowered.

Clinical picture.—Eczema may be present in both breast-fed and artificially-fed infants and is *common during the dentition period*, i.e. between 6 months and 2 years, but most common between 6 months and a year. In some cases, however, it appears in the first few weeks of life and persists off and on for some years. The extent varies enormously. The usual site of the lesion is on the *cheeks and forehead*, but almost the whole of the body may be involved. The buttocks and flexor surfaces of the arms and legs are other common sites. In some children only small patches in the bend of the elbow and behind the knees, on the cheek or behind the ears ever develop, waxing and waning.

There are two types of eczema, the *dry or scaly* and the *weeping* type. Various authorities maintain that in breast-fed infants one or the other of these types is the more common. There is, however, no way of making such a distinction. The dry type may weep, and the weeping variety may, with care, recede to the dry type and then clear up.

Irritation.—Nothing is so irritating to the infant as eczematous patches on the face and limbs. The child lies rolling the head from side to side, attempting to rub the cheeks into the pillow. Meanwhile the legs are scratched one on the other, and the hands, if free, tear at the cheeks and face or the legs. This is aggravated by warm weather, or by overclothing the infant. As a rule the child is inclined to be *large, over weight and fair* and the skin thin, soft and fine. The history shows a rapid gain in weight and there is a tendency to catarrh of the bowel with loose motions. *With diarrhœa the eczema improves, with cessation of the loose stools it is accentuated.*

Treatment.—The most important thing is the *prevention of scratching and irritation* of the affected parts. The arms should be encased in cardboard splints, and if necessary a loop of bandage should be pinned to the sleeve, fastening the arms away from the sides to the cot or perambulator.

Local applications.—If the face is affected and if it is the dry type of eczema, the lesion should be kept dry with calamine lotion, or if it tends to weep slightly an ointment such as ung. resorcini (resorcin, 10 gr.; hydrous wool-fat, 2 dr.; soft paraffin to 1 oz.) should be applied. If there are crusts or scabs nothing is so useful as starch poultices. A tablespoonful of starch should be mixed to a paste with cold water, and to this sufficient boiling water should be added to dilute the whole to a thin jelly. This should be spread on lint, covered with butter-muslin and applied with the muslin next the affected part, the whole being covered with a thick layer of cotton-wool and bandaged on. These poultices may require to be applied on a mask,

holes being cut for the eyes, nose and mouth (Fig. 68).

Diet.—There can be little doubt that the sooner the infant is put on a mixed diet, replacing milk foods by solids, the sooner the eczema clears up; in other words, exclusively bottle-fed infants, if put on a diet with cereals and soups, are much better and the lesions settle more quickly. It has been found that taking the child off fresh milk altogether, and putting him on to

Fig. 68.—Eczema, showing method of applying dressing to face, and cardboard to arms.

a synthetic milk such as Almata, brings about a great improvement, but this has not been the author's experience. In breast-fed children the quantity should be reduced, or a food such as Benger's, made with skimmed milk, should be substituted for some of the breast-milk, to prevent gross overfeeding. Taken at the very start and treated vigorously, eczema can be kept well in hand, but once the lesions have become general the problem is extremely difficult, and the

assistance of a district or hospital-trained nurse in applying the dressings becomes almost a necessity.

Medicinal treatment.—A little chloral and bromide should be given to the infant if extremely restless.

Management.—The clothing should at all times be light and it is best to have silk material next the skin. In bathing the child, avoid soap when the skin lesions are active, substituting toilet oatmeal.

Prognosis.—The mother of a child with eczema may be assured that scars will not be left on the skin from patches of eczema, in fact the skin remains singularly clear and soft afterwards. The great majority of cases of eczema clear up within a year or 18 months.

Pityriasis Rosea

This eruption begins as a solitary pink scaly patch on the neck, chest, abdomen or limbs, and after a few days smaller patches may appear all over the trunk. They are often the size of a finger-nail or larger. The whole eruption lasts about six weeks and then fades. Often it is accompanied by a little fever. The *diagnosis* can be made by recognizing that the herald patch precedes the general eruption by a week or ten days. It can be differentiated from ringworm by an examination of the scales for the fungus. For *treatment* apply salicyclic acid ointment, 15 grains to the ounce.

Psoriasis

This is more common after the age of 5 or 6 years and consists of small, round patches of a dull-red colour, covered with silvery scales. It is more commonly found on the knees, trunk and elbows, the patches varying from the size of a sixpence to that of a shilling. Usually there is such a silvery appearance to the lesion that the diagnosis is easily made. The disease is often hereditary, and in some cases it may come and go.

The *treatment* consists in the local application of liquor picis carbonis scrubbed into the lesion with a stiff brush. An ointment such as ung. oleum cadini (B.P.) is also useful. Giving a little arsenic also often expedites the disappearance of the lesion.

CHAPTER XVI

INFECTIOUS DISEASES

Infectious disease plays an extremely important part among the diseases of childhood, and assumes special importance during the child's school life. In former times the children of even the well-to-do had had some, if not most, of the infectious diseases before the school age. Now, due to immunization, the care of contacts, observance of quarantine and, among other factors, better ventilation and hygiene, children may reach the secondary or public school having had only one or two of these. This has introduced a new problem into school life among the older children. There is no doubt in the author's mind that putting off infectious disease until a child is older and stronger is a very great advantage. The mortality of most acute infectious fevers is much higher among children of the pre-school age than among those above it. This is borne out in the following statistics : [1]

	Mortality	
	Under 5 years.	*Over 5 years.*
Measles	11 per cent.	1 per cent.
Scarlet fever	8 „ „	1 „ „
Whooping-cough	14 „ „	1 „ „

An excellent summary of the position in regard to infection and the period of quarantine necessary in various diseases is given in a leaflet issued by the Medical Officers of Schools Association and here reproduced.

[1] J. D. Rolleston, "Acute Infectious Fevers" (Heinemann, 1929).

Tables and Rules issued by the Medical Officers of Schools Association

Measles, Rubella,* Mumps, may be infectious	4 days	before the appearance of the rash or other symptoms.
Chicken-pox " " "	2 days	

Whooping-cough may be infectious at the first symptoms of catarrh.

A Boy may Return to School after Exposure to Infection of

Measles	when 16	clear days have elapsed from the last date of exposure to infection.
Rubella,* Chicken-pox	" 20	
Whooping-cough	" 21	
Mumps	" 24	

Provided disinfection of clothes is carried out at commencement of quarantine.

A Boy who has Suffered from an Infectious Disease may Return:
Rubella*.—10 days from the appearance of the rash.
Measles.—14 days from the appearance of the rash.
Chicken-pox.—When all scabs have fallen off.
Mumps.—Seven days after all swelling has disappeared, but not earlier than 14 days from the beginning.
Whooping-cough.—Five weeks from beginning, if whooping has ceased 14 days.
Scarlet fever.—When certified free of infection.

Provided patient and clothes have been disinfected.

Scarlet Fever

Incubation period.—This is from 2 to 6 days, but contact cases should be quarantined for 10 days to be quite safe. A case of scarlet fever should be isolated for from 5 to 6 weeks, or until all discharges from the ears and nose have cleared up.

Mode of infection.—This is either directly from some infected individual to a healthy person, or through the agency of milk. It is said that it can be transmitted on some article of clothing or in a book (fomites), but this view is open to question.

* German measles, epidemic roseola.

Seasonal incidence.—Scarlet fever is most prevalent in the late summer and early autumn, but may occur in epidemic form at any time of the year.

Age-incidence.—It is uncommon in infants under one year, and most common between 5 and 9 years. Sporadic cases, where no infective source can be traced, are frequently reported, and it must be concluded that there are a large number of scarlet-fever "carriers."

Etiology.—Recent work has shown that scarlet fever is due to a specific streptococcus, which generally attacks the throat first.

Diagnosis.—The diagnosis may sometimes be extremely difficult, as it depends on the rash, which can be simulated by enema rash, by toxæmias or by food-poisoning. The sore throat can also be simulated, and probably the most important feature in the diagnosis is the peeling which occurs afterwards.

The **Schultz-Charlton test**, which is performed by injecting intradermally 0.2 c.c. of a 1-in-10 dilution of scarlatinal antistreptococcus horse serum, is an excellent means of diagnosis. The serum is injected into the arm of patients exhibiting a rash, and where the rash is due to scarlet fever there is a blanching about the point of injection 4 to 10 hours later, which persists for from 12 to 72 hours.

Symptoms and clinical picture.—In very mild cases which are often called *scarlatina*, beyond a very slight sore throat and a rise of temperature, with a transient rash, nothing may be seen, the whole illness subsiding in 3 or 4 days and ending in complete recovery. In the more severe cases there is an abrupt onset, usually with vomiting and both headache and intense sore throat. The child is ill and dull. The pulse and respiration-rate are increased, the skin is hot, and the cheeks are flushed. The rash appears in from 24 to 48 hours, but occasionally does not appear until the third or fourth day. In some cases, however, it occurs first of all. There is usually only one vomit.

The rash.—This is erythematous and comes out on the trunk and limbs (except the palms of the hands

and soles of the feet), and on the neck towards the lower borders of the jaw, where it merges with the flush on the face. Round about the mouth there is a pale area, the so-called "circumoral pallor." The rash may last for a few hours or as long as a week.

Desquamation.—At the end of a week to 10 days the skin begins to peel, starting round the neck and upper part of the chest, then becoming general. The skin of the hands and feet peels last, and tags of skin can be seen for some time afterwards at the roots of the nails. The peeling is generally in proportion to the intensity of the rash and lasts for from 4 to 8 weeks.

The tongue.—The so-called "strawberry tongue" can be seen on the first or second day—a white tongue with red papillæ showing through. As the disease progresses the tongue becomes red all over, the throat being painful and the tonsils enlarged and fiery red, with enlarged glands at the angle of the jaw.

Complications.—Hæmorrhagic nephritis is one of the most common complications. Occasionally parenchymatous nephritis follows. Cervical adenitis and ulcerative stomatitis come next in order of frequency, and rheumatic carditis and otitis media are also found.

Prognosis.—In fatal cases where the infection has been especially virulent death is due to a general septicæmia, with an accompanying myocarditis, or to some complication such as pneumonia or nephritis. Generally speaking the prognosis as to life is excellent, the mortality being extremely small, but whereas for ages under two it is about 15 per cent., for ages over two it is less than 1 per cent.

Treatment.—The patient is nursed in bed from the beginning. The diet should be the fever diet described on p. 415. If the temperature is unduly high, above 103, tepid sponging should be resorted to, with water at about 90° F. A little aspirin (2½ grains), or sodium salicylate (5 grains) and potassium chlorate (3 grains) is extremely useful. The throat may be swabbed with weak iodine or perchloride of iron or perchloride of mercury. The administration of quantities of alkali is specially indi-

cated, and from 30 to 60 grains of bicarbonate or citrate of soda should be given daily during the febrile period. Bland drinks, including much water, should be insisted on. The greatest possible care should be taken of the mouth to prevent stomatitis, and it should be swabbed every two or three hours during the day with borax-and-honey, glyco-thymoline, peroxide of hydrogen or permanganate of potash. If there is any suggestion of involvement of the heart, the patient should be kept lying perfectly flat for a prolonged period. A careful examination of the urine should be made for albumin daily. A watch must be kept on the ears for any signs of otitis media.

Serum.—Anti-scarlet-fever serum (antistreptococcus serum) may be given as a prophylactic. In most cases 5 c.c. is sufficient to protect "contacts." As treatment, 5 to 10 c.c. of the concentrated anti-scarlet-fever serum (Burroughs, Wellcome & Co.) is said to give excellent results, but, apparently, in the most severe cases the best results are obtained by giving repeated large doses. In the average mild case it does not appear to be essential.

The Dick test.—This is performed by injecting intradermally 0.2 c.c. of a 1-in-1000 dilution of the filtrate obtained from a broth-culture of the streptococcus of scarlet fever. A control is also used. If the patient is susceptible to scarlet fever a reddened area appears in from 6 to 12 hours round about the injection. This test is of considerable value, as the "susceptibles" can be immunized in any given epidemic.

Rubella (German Measles)

Age-incidence.—This disease may attack children at any age, but it is more common after the age of five. It is spread by direct contact from child to child. Rubella is not to be classed among the most common infectious diseases, and it is only feebly infectious.

Incubation period.—This is from 10 to 21 days, but it is usually 15 or 16 days, the child being infectious 4 days before the appearance of the rash or other

symptoms. He may return to school when 20 clear days have elapsed from the last date of exposure to infection, but if he has been himself infected, he cannot return until 10 days after the appearance of the rash.

Clinical picture and symptoms.—The disease commences with very slight constitutional symptoms, the temperature rising as the rash comes out. There may be coryza and sore throat, and there is usually enlargement of the glands posterior to the sternomastoid, and round about the occipital and mastoid region. As a rule, however, the rash is the first indication that the child has become infected. Occasionally conjunctivitis (pink-eye) accompanies the disease. There are few or no complications.

The rash.—The distribution of the rash is on the face and scalp, trunk and extremities. It may appear somewhat like an urticaria, or a diffuse erythema like that of scarlet fever. It rarely lasts longer than three days and has often completely disappeared in 24 hours. As the rash tends to be a measly one, the disease is usually mistaken for measles, but on the other hand it may be mistaken for an urticaria or nettle-rash, due to some toxæmia or drug. It should be borne in mind that in measles, unlike rubella, two or three days before the appearance of the rash there is some indisposition and coryza.

Treatment.—The child should be kept in bed until the temperature has become normal, and should be isolated for one week after the appearance of the rash. No other treatment is necessary.

The mortality from this disease is negligible.

Measles (Morbilli)

Incubation period.—This is from 7 to 21 days, the child being infectious 4 days before the appearance of the rash or other symptoms. A child may return to school when 16 clear days have elapsed from the last date of exposure to infection, but if he has been infected himself, he cannot return until 14 days after the appearance of the rash. The etiology is unknown.

Clinical picture.—Four or five days before the rash appears the temperature rises to 100° or 101°F. and remains up for 48 hours, then falls, the child feeling quite well. During this febrile stage the eyes and nose run, and the face is puffy. The child looks heavy and appears to have a bad cold. On examination the inside of the mouth and cheeks shows Koplik's spots, and laryngitis or croup may be present at this stage. On the fourth day from the commencement the temperature, which is as a rule normal, suddenly rises again and the rash appears behind the ears and round about the mouth, and then spreads rapidly over the rest of the body. The glands, which have been slightly enlarged from the beginning, become prominent, especially behind the ears and posterior to the sternomastoid. The throat is sore and a little cough may develop, with catarrhal bronchitis. The temperature remains up for from one to five days or longer. There is a degree of conjunctivitis, so that the eyes are sore and tend to discharge.

Koplik's spots.—These are seen on the first, second or third day and are best seen inside the cheeks, especially towards the posterior part of the mouth. They are small, about the size of a pin-head, raised, and bluish white in colour, surrounded by a red areola. Their absence, unfortunately, does not in any way exclude a diagnosis of measles.

Rash.—The rash of measles is characteristic. It starts as small pink macules which rapidly form larger irregular-shaped patches or blotches. Large areas may become involved, giving an erythematous appearance. This rash may last for from 3 to 5 days, and often leaves a faint brown, stained appearance behind it. The temperature falls with the fading of the rash. Prodromal rashes are not uncommon, appearing during the period of incubation. They are usually blotchy and erythematous in type.

Complications.—The most common of these are pneumonia, otitis media, conjunctivitis and stomatitis. An extreme rarity is measles encephalitis, the mortality

from which is high, but when recovery takes place it is complete. Repeated lumbar puncture and convalescent measles serum subcutaneously are indicated.

The *mortality* under the age of five is about 11 per cent., whereas above five it is negligible.

Treatment.—The child should be put to bed in a darkened room, the eyes being thus rested. The diet should be the fever diet (*see* p. 415) with an abundance of bland fluids, and cereals and other carbohydrates should be pushed on with. The bowels must be kept well open and some alkaline saline, such as milk of magnesia or Eno's Fruit Salts, should be given. A mixture of sodium salicylate (5 grains) and potassium chlorate (3 grains) should be given three times daily. The child should be kept in bed until the temperature has been normal for one week. The greatest care should be taken of the mouth, swabbing it out two or three times daily with peroxide of hydrogen, glyco-thymoline or glycerin and borax.

The treatment of the disease by means of serum from a convalescent patient has not been very satisfactory, and in our present state of knowledge is not advisable. As a prophylactic, however, there is no doubt that from 3 to 5 c.c. given to contacts as early as possible during the incubation period either completely aborts or greatly attenuates the disease.[1]

Mumps (Epidemic Parotitis)

Mumps is an infection of the parotid gland, usually bilateral. It may occur at any age, but is most common between the ages of 5 and 15, and is conveyed directly from one child to another. The disease is due to a filter-passing micro-organism, and this organism is present in the patient's saliva.

Incubation period.—This is from 14 to 21 days, and the child may be infectious 4 days before the appearance of the symptoms. He may return to school

[1] D. N. Nabarro and A. Signy, "The Prophylactic Treatment of Measles," *Lancet*, 1929, i. 1106.

when 24 clear days have elapsed from the last date of exposure to infection, but if he has been infected himself, he must not return until 7 days after all swelling has disappeared, and not earlier than 14 days from the beginning.

Clinical picture and symptoms.—The disease commences with pain in the region of one parotid gland, which begins to swell, the pain being more marked on opening or shutting the mouth. At the same time there is some degree of fever. The sub-maxillary and sub-lingual glands are often involved in addition. After 4 or 5 days, the other parotid gland becomes involved, the first one rapidly subsiding. In a mild case both swellings have subsided in from 10 days to a fortnight. In more severe cases the child is very ill, with a high temperature and a dirty tongue, unable to open the mouth sufficiently to take food other than liquids. The blood shows an increase of the lymphocytes.

Complications.—*Orchitis* is the commonest complication, and the most severe, the testicles becoming greatly swollen. There is a danger afterwards, when the swelling has subsided, of atrophy or destruction of these organs. In the female the vulva and breasts may be involved, and also the ovaries, which sometimes are destroyed. Gangrene of the vulva is rare. Occasionally acute pancreatitis occurs. A rare complication is *mumps meningitis*. On the whole the outlook is good, and usually mumps is to be considered a mild disease.

Treatment.—The child should be confined to bed on a fever diet (*see* p. 415). Chlorate of potash (3 grains) and salicylate of soda (5 grains) are most efficacious. The greatest care should be taken to keep the mouth quite clean.

Whooping-cough (Pertussis)

This is one of the most common diseases of childhood, and in infants is an extremely fatal one. In older children it is comparatively mild.

Seasonal incidence.—The number of cases is greatest during the first quarter of the year.

Age-incidence.—The patients are usually round about the age of two. Very often whooping-cough is associated with measles, but the relationship between the two diseases is not clear.

Incubation period.—This is usually about 14 days, but may vary from 7 to 21 days. Whooping-cough is infectious as soon as the first symptoms of catarrh appear. A child may return to school when 21 clear days have elapsed from the last date of exposure to infection, but if he has been infected himself he may not return for 5 weeks from the beginning, or until whooping has ceased for at least 14 days.

Bacteriology and pathology.—It is generally supposed that whooping-cough is due to the Gram-negative Bordet bacillus, which is certainly found in the sputum early in the disease. The infection is essentially a tracheitis, much semi-purulent material mixed with mucus being produced. The cause of the whoop is not known, but it may be due to a secondary enlargement of the tracheal glands, pressing on the trachea or larger bronchi. A help in the diagnosis may be found in the blood-count, where a distinct lymphocytosis occurs. The count may rise from 20,000 to 30,000, with a proportion of 60 per cent. or 70 per cent. of lymphocytes. In the author's opinion the blood-count is seldom helpful and definite enough in those cases where the diagnosis would be of most value. Culturing the fresh sputum on special media for the Bordet bacillus is highly recommended in doubtful cases.

Onset and clinical picture.—Whooping-cough should be divided into four stages of a fortnight each.

First stage.—This is the period after contact when the child is incubating the disease. During this period as a rule no symptoms are noted.

Second stage.—During this fortnight there is a tendency for an increased catarrh of all the mucous membranes. The child ceases to gain weight, and has a tendency to run a slight temperature. A persistent

hacking cough appears and gradually becomes worse, especially at night. The nose runs and the face is puffy, and a little albumin may appear in the urine. Towards the end of this fortnight the cough becomes steadily more and more spasmodic, that is, it occurs in sudden severe paroxysms, followed by a comparatively long period quite free of the cough. The child is wakened or disturbed more and more frequently during the night. A small ulcer may appear on the tongue where the frenum impinges on the lower incisors. This is most frequently seen in children about 2 years of age.

Third stage.—At about this time, or just previous to this stage, whooping-cough is strongly suspected or has been diagnosed, as the child commences to whoop and vomit. Each paroxysm of coughing ends in such a spasm that on inspiration a long-drawn-out whoop may be heard, the face becoming cyanosed, the eyes bloodshot and the child running to be held until the paroxysm is over. Along with the bout of coughing, the last food taken may be mechanically vomited. A typical whoop may be said to consist of a series of short expiratory coughs, with a long inspiratory whoop, which continues until a plug of mucus is dislodged, when the spasm ceases. Mucus and saliva stream from the nose and mouth during the attack.

Fourth stage.—During this last fortnight the whole process rapidly retrogresses. The whoops become less frequent at night, so that the child's sleep is less disturbed. During the day there is less vomiting and consequently the child begins to gain weight. By the end of this period in the average case the cough has practically ceased.

Recurrences.—In some cases there is a continuance of the whoop, and bouts of coughing occur for many months. There seems no explanation of such cases, except that the child has acquired a habit or trick of whooping. Other children, who seem to recover completely, develop a slight pulmonary catarrh or bronchitis some months later, with a return of what appears to be a typical whoop.

Prognosis.—Among children under five the mortality is about 14 per cent., whereas over five it is less than 1 per cent.

Complications.—Undoubtedly one of the most distressing complications is **convulsions.** At the end of a bout of coughing the child, now extremely cyanosed, passes rapidly into a convulsion, which continues for some minutes. Such cases on the whole do extremely badly, and there appears to be a tendency for minute cerebral hæmorrhages to occur at this time. Certainly cases of hemiplegia are not uncommon after whooping-cough convulsions. In one case seen recently by the author, the child was left physically normal but mentally most unstable and excitable, the picture one might expect following encephalitis.

Epistaxis is another quite common complication. It should be treated by keeping the child in the upright position, preferably with the arms above the head, and by applying cold compresses.

Wasting, due to vomiting and accompanying anorexia, is also a severe and common complication. This may become a real problem in certain cases. It is advisable during the stage when vomiting is most frequent either to feed the child again immediately the vomit has occurred, or to give frequent small feeds of thin fluids containing glucose, which can rapidly pass out of the stomach.

Miliary tuberculosis, engrafted on or following whooping-cough, is a common occurrence, and should be suspected where the whooping-cough fails to subside, or where the pulmonary complications are severe.

Broncho-pneumonia.—This is probably the most serious and frequent complication of whooping-cough. In these circumstances the term secondary pneumonia is employed. The prognosis in such cases is not good, as there is a tendency for such pneumonias to remain unresolved, leading to pulmonary fibrosis or bronchiectasis. Emphysema may also develop.

Treatment.—During the acute febrile stage, that is in the second stage, when the cough is daily increasing,

the child should be indoors and in bed if the weather is inclement. During the summer months, he should be nursed in bed before a widely-open window. Once the temperature has become normal, the child is best treated out of doors, getting about freely, unless there is accompanying bronchitis. In a large series treated by the author[1] the complications were shown to be much fewer in children who were up and about than in children confined indoors.

Tonics.—Cod-liver oil and malt, cod-liver oil emulsion, or one of the cod-liver oil preparations is strongly indicated. If the child tires of these, malt and iron should be given.

Medicinal treatment.—Undoubtedly the most satisfactory mixture is one containing belladonna, which tends to keep the secretions dried up, and potassium bromide, which acts as a mild sedative. The belladonna is also indicated as an antispasmodic. As a rule a mixture containing tinct. bellad. (5 minims) and pot. brom. (3 grains) should be given four to six times daily. As an alternative, chloretone in doses of from 2 to 4 grains, four times daily, is a beneficial sedative.

Vaccines.[1]—In the hands of the author vaccines employed in treatment have not proved efficient, but as a prophylactic measure they are well worth giving.

Other treatment.—The practice of giving ether injections and deep X-rays to the mediastinum is not to be recommended. Many drugs, such as turpentine, creosote and garlic, have been prescribed, but in the author's hands have met with no success.

Diet.—During the early stage a light diet, containing a preponderance of starchy cereal foods, should be given. For instance, a child of three should be given a little cereal, with occasionally an egg or fish for breakfast; gravy, potato, vegetables and stewed fruit at midday; and bread-and-butter and honey, milk and sponge-cake for tea. After the second stage, when the child is up and about, despite the whooping and

[1] Donald Paterson and J. M. Smellie, "The Value of Vaccines in the Treatment of Whooping-cough," *Brit. Med. Journ.*, May 6, 1922.

vomiting, the diet should be as liberal as the child will take and digest. Immediately after a whoop and vomit the child should be given more food if he will accept it.

Chicken-pox (Varicella)

Etiology.—Chicken-pox appears to be caused by a filter-passing virus.

Incubation period.—This is from 12 to 20 days, the child being infectious for two days before the vesicles appear. A child may return to school when 20 clear days have elapsed from the last date of exposure to infection, but if he has been infected himself, he must wait until all the scabs have fallen off.

Symptoms and clinical picture.—Chicken-pox is one of the mildest diseases of childhood. As a rule the first signs of infection are the vesicles, but the child may have been out of sorts for a day or two previously. The rash appears as a series of red papules, which rapidly become vesicles or blisters.

Distribution of rash.—The rash appears first on the trunk, face and scalp, then on the limbs, sometimes in the mouth. It is extremely itchy.

Fever develops with the appearance of the rash, but the temperature falls to normal within a day or two. Successive crops of the eruption may appear for several days, and pocks may be seen alongside one another in different stages of development.

Complications and treatment.—Among the commonest complications are tonsillitis, tracheitis and laryngitis. These should be treated by keeping the patient quietly in bed and giving inhalations of friars' balsam, which will give relief. Salicylate of soda (5 grains) and chlorate of potash (3 grains) should be given for the tendency to sore throat. Gradually the scabs fall off, and with the disappearance of the last, which are usually among the hair, the child should be considered free from infection. Apart from this there is no treatment, except to prevent the child from scratching and thus causing scars.

Diphtheria

Etiology.—Diphtheria is caused by infection with the Klebs-Loeffler bacillus. It is most common in the autumn and winter months.

Incubation period.—This varies from 1 to 7 days. The common sites of infection are the tonsils and palate, nose and larynx, ears, anus and vulva. The disease is usually transmitted by carriers or through milk.

Age.—At birth children are immune, and most adults also are immune. The period of greatest susceptibility is from 1 to 10 years of age, as at the age of about one year the child loses the immunity with which it was born.

Symptoms and clinical picture.—The symptoms are produced by the toxins of the germ. The organism itself is localized on the surface of the tonsil or other part of the body, the toxins are absorbed. As a rule the temperature is slight, but occasionally may be high, especially in later stages. Sore throat is rarely complained of by patients of less than seven. The cervical glands are enlarged in most cases of faucial diphtheria, and very often there is a little vomiting. The urine contains albumin, the tongue is dirty, the breath offensive and fœtid, the child is drowsy and pale and shows the signs of poisoning. If untreated, a proportion of the patients die of heart-failure, resulting from toxic myocarditis, or the infection spreads to the trachea, causing death from suffocation.

Diagnosis.—In every case throat swabs should be taken, even where the disease is not seriously suspected. Acute follicular tonsillitis can be differentiated because of the flushed face and high temperature, with a feeling of extreme malaise. In diphtheria, as we have said, the temperature is often only slightly elevated, and in the early stages the child does not feel particularly ill. The patches on the throat in diphtheria are grey and raised, and when an attempt is made to swab them off they are adherent and bleed. There is, as a rule, only one patch on each tonsil, whereas in follicular tonsillitis

many small grey areas are to be seen. Chronic discharge from the ear or from the nose should always be swabbed to exclude the presence of the Klebs-Loeffler bacillus, as these are not uncommon sites for the infection.

Prognosis.—Under one year the mortality appears to be about 44 per cent., and this gradually decreases until by the age of 15 years the mortality is practically negligible. The mortality for all ages would appear to be about 8 per cent.

Treatment.—The treatment of the disease in the early stages is extremely satisfactory, as we have a specific in the antitoxin serum which can always be relied upon. For ordinary very slight infections 4,000 to 8,000 units should be considered the minimum, and 20,000 units should be regarded as a moderate dose. In severe infections doses up to 100,000 units are commonly given. The antitoxin is best given subcutaneously, beneath the abdominal wall. The child should be kept lying flat because of the danger of heart-failure due to a damaged myocardium. In the recumbent position as little work as possible is thrown on the heart. The greatest care should be taken of the mouth, which should be carefully swabbed out with mild antiseptics to prevent infection. The diet should be the fever diet (*see* p. 415) and should be pressed on with, giving the maximum that the child is able to digest.

Complications.—Cases which have been extremely slight, or have not been diagnosed, often give rise to post-diphtheritic paralysis (for the symptoms of which *see* p. 312) in from three to six weeks. Palatal paralysis occurs, the food being regurgitated through the nose. The knee-jerks are lost and there is weakness of the limbs, squint and difficulty in reading.

Nasal diphtheria.—In these cases serum may be given, though no great help can be expected from it. Spraying the nose with the serum has been recommended, but local application of antiseptics, syringing with glyco-thymoline or Condy's Fluid, or the application of argyrol drops (5 per cent.) is to be preferred.

In a certain proportion of cases the infection disappears on removal of the infected adenoids by operation. This measure is not only justified but is indicated.

The Schick test.—This is a test which makes it possible to determine whether an individual is susceptible or immune to diphtheria. It is performed by injecting 0.2 c.c. of a diluted diphtheria toxin into the skin of the forearm (intracutaneously). A control of heated toxin is used on the opposite arm. If the patient is not immune to diphtheria a reaction appears round about the injection, indicating that he is susceptible and should therefore be immunized by means of toxoid antitoxin serum. If the result is negative nothing further need be done.

Immunization by toxoid antitoxin serum.—Injection of this serum produces an active immunization. It takes some weeks to develop, and for practical purposes produces an absolute immunity against infection by diphtheria for life. The best method appears to be the injection of from ½ to 1 c.c. of the toxoid antitoxin serum at weekly intervals for three weeks. After say three months the Schick test can be repeated and will as a rule be found negative. If positive, a further course of the serum should be given to increase the immunity.

There appears to be no contra-indication to this method of immunization against diphtheria, and as the toxoid antitoxin seems a most stable product, which keeps perfectly well up to eighteen months after manufacture, there is no difficulty in obtaining the product nor need there be any fear in using it.

Typhoid Fever (Enteric Fever)

Etiology.—This disease is caused by the Bacillus typhosus, a Gram-negative and motile organism. The organism is killed by boiling or by carbolic acid in a strength of 1-in-20.

Mode of infection.—As a rule the patient is infected by means of water, milk or some milk product such as

ice-cream, or by direct contact with a typhoid patient or "carrier." Outbreaks in institutions have occasionally been traced to the presence of a "carrier" in the kitchen. Ice and salads have both been found infected, the latter from contaminated soil.

Clinical picture and diagnosis.—Generally, the onset of the attack is gradual, with a slight headache, epistaxis and a staircase-like rise of temperature. In a moderately severe case there is a gradual onset of delirium and drowsiness, followed by extreme prostration and toxæmia. On the other hand the disease may be ushered in by acute diarrhœa and vomiting. The period of continuous fever may vary from a week or 10 days to 6 or 10 weeks. An average case in childhood lasts 3 or 4 weeks. There is almost invariably some intestinal disturbance, either diarrhœa and vomiting at the beginning, or later on some degree of looseness of the bowels (pea-soup stools). Occasionally, however, there is obstinate constipation.

The spleen.—This is found to be enlarged in the vast majority of cases during the first few days of the illness, a sign which should always be sought for.

The blood.—The white count shows a normal number of white cells or an actual leucopenia, such as say 6,000 white cells, 60 per cent. being polymorphs and the remainder lymphocytes.

Agglutination (Widal) test.—This is seldom positive before the tenth day and reaches its maximum about the sixteenth day. It should not be considered a positive test unless it agglutinates the typhoid organism in a dilution of 1 in 25. As a rule the test gives a positive agglutination in a dilution of from 1 in 250 to 1 in 1000. The author has very occasionally known true cases of typhoid which gave a negative agglutination test.

Cultures of the blood and urine may be found positive during the first 4 or 5 days of the illness.

Pulse-rate.—In a classical case of typhoid the pulse is slow and bounding, but this cannot be relied upon.

Rash.—The so-called "rose spots" are diagnostic of this condition and appear at the beginning of the

second week, usually over the abdomen, chest and back. The spots, which are round and are about the size of split peas or smaller, disappear on pressure. They are few in number, usually not more than 4 or 6 being present at a time.

Headache.—This symptom is as a rule very severe and, coupled with epistaxis, is most suggestive.

Wasting.—One of the most alarming features of typhoid fever is the tendency to wasting. A limited quantity of food only can be administered and digested, whereas with a high continuous temperature, restlessness and delirium, the caloric requirements of the patient are extraordinarily high, and consequently severe wasting almost invariably occurs.

Gradually, in the third or fourth week the temperature settles, the tongue becomes clean, the appetite improves, the delirium ceases, and the patient begins to recover.

Pathology.—During the first three or four days typhoid fever is a blood infection and there is an actual septicæmia. It is possible during this stage to recover the organism from the blood and also from the urine. After this time the infection appears to settle in the bowel, especially in the lymphoid tissue, causing hyperplasia and ulceration of the Peyer's patches and swelling of the mesenteric glands and of the spleen. Evidence of the toxicity of the infection, however, is shown by cloudy swelling throughout all the organs, and there may be a severe peripheral neuritis or encephalitis as a result of these toxins. The Peyer's patches slough and separate, leaving shallow ulcers behind which tend to perforate. The sloughs may show a tendency to hæmorrhage.

Prognosis.—In children the mortality is extremely low, perforation being almost entirely unknown and hæmorrhages exceedingly rare. Figures given by J. D. Rolleston in "Acute Infectious Fevers" show that the mortality under five years is 8 per cent., from five to ten years about 6 per cent., and from ten to fifteen years 7 per cent. This rate is much higher than in a series of cases seen by the author.

Treatment.—For the first few days as a rule the case is not diagnosed. Probably, during this time, in view of the gradually rising temperature, drowsiness, and vomiting or diarrhœa, the usual treatment for acute gastro-enteritis has been administered. The child will have been given a dose of castor oil and put on a very light diet. Once the diagnosis is made, however, the nursing and treatment should be systematized along the following lines:

The patient should be given an abundance of sugar-water or fruit juice and water, and fed regularly on diluted milk thickened with some starchy preparation, such as Benger's Food, groats, cornflour, arrow-root, or broth thickened with potato. If curds appear in the stool the milk should be peptonized. As large a quantity of the starchy milk food should be given as the patient is able to digest. When the temperature begins to subside, more and more solid food should be pressed on with, although it is at this time that the sloughs begin to separate and there is fear of perforation. Despite this, potato and gravy, a little raw steak and eggs in various forms should be given. If there should be any tendency to constipation some liquid paraffin preparation should be employed to keep the motion soft and prevent damage or straining on the weakened bowel.

The *nursing* is of the utmost importance. During the acute stages iced drinks or tepid sponging may help to prevent hyperpyrexia. A little aspirin is also of value. The greatest possible care should be taken of the mouth in order to prevent ulcer-formation, and several times daily it should be swabbed out with peroxide of hydrogen, glyco-thymoline, or borax and glycerin. A water-bed is desirable to prevent bed-sores, and all the pressure points should be sponged over daily with some hardening lotion such as methylated spirits or alcohol. All excreta should be collected and dealt with by disinfecting with 1-in-20 carbolic acid. Where hæmorrhage is suspected in view of rapidity of the pulse, blanching and collapse, absolute rest should be imposed with an ice-bag placed on the

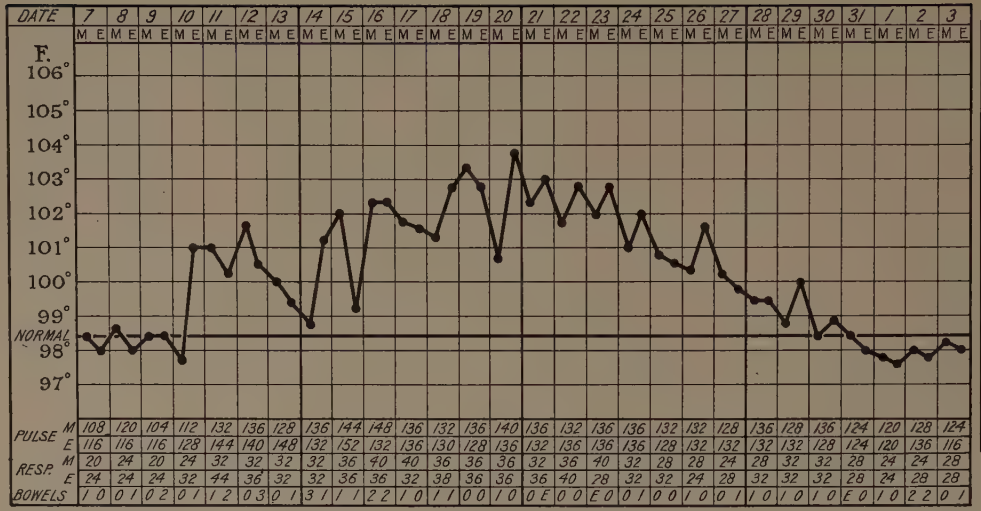

Fig. 69.– Complete temperature chart of a case of paratyphoid B in a child aged 6. Note that the pulse-rate ranges from 100 to 148, and that at no time did the child have diarrhœa.

abdomen. The patient should be kept carefully under observation in view of the possibility of a laparotomy being necessary. Where perforation is suspected on account of a sudden rise of temperature with abdominal pain and tenderness and obstruction, laparotomy should be undertaken at once.

Complications.—Complications of typhoid fever in children are exceedingly rare, and of these the two most important have already been mentioned, namely, perforation of the bowel and hæmorrhage from the bowel. Probably the commonest complication is ulceration of the mouth and tongue. Another takes the form of abscesses which may be present in any bone or joint, one of the most common sites being the head of the tibia. Polyneuritis of the toxic variety, a prolonged complication, occasionally accompanied by encephalitis, is rare.

Paratyphoid Fever

This is much more prevalent in London than typhoid fever, and most of the epidemics of recent years have been of this variety. Paratyphoid has a clinical picture like that of a very mild form of typhoid. The duration of the fever is shorter, being from two to three weeks at most (Fig. 69). The feeling of malaise and general upset may be severe, but is usually mild or even slight. The organisms responsible are the Bacillus paratyphosus A and B, and these can either be demonstrated on culture of the blood or urine or later the positive agglutination (Widal) test is given, thus confirming the diagnosis. The prognosis and the treatment are the same as in typhoid fever.

Influenza

Influenza may be either of the epidemic type, in which event a large proportion of the population is affected, or of a sporadic nature. In the great pandemic of 1918 the proportion of infants and children affected was extremely small. The infection may show such a great variety of clinical features that the diagnosis is

always of the greatest importance. Because of the difficulty in diagnosis the term influenza is often used and later found to be misapplied.

Etiology and pathology.—The accepted view is that influenza is due to Pfeiffer's bacillus, a Gram-negative organism. Certain authorities, however, claim that this disease is caused by some filter-passing virus, its powers of infectivity being extremely great. Certainly, when found in lesions of the lung Pfeiffer's bacillus is usually accompanied by streptococci and pneumococci. The chief damage to the body generally appears to be toxic, and cloudy swelling of all the organs is found at post-mortem. Pure cultures of Pfeiffer's bacillus have been grown both from the cerebro-spinal fluid and by blood culture.

Clinical picture.—This may show great variations both with the particular epidemic and from year to year. Usually the onset is acute, the temperature rising suddenly to 102° to 103°, with aching all over, a disinclination for food and a tendency to vomit. There may be a slight coryza or sore throat and the tongue is dry and coated. Mild cases subside in 2 or 3 days, but the more severe cases may last for a week or longer. In addition to a general infection, there is also, it is believed, a septicæmia. Each epidemic may attack some particular system and there is a preponderance of symptoms in that part of the body. Thus in 1929 the epidemic, beginning in Canada and the United States, reached the British Isles early in the year. This outbreak was of a particular kind which, besides the usual general symptoms of influenza, brought with it a strong tendency to *otitis media* and its complications. The *respiratory system* also may be specially affected, with mild or severe catarrh of the upper respiratory tract, in some cases extending down the larger to the smaller tubes, giving rise to bronchitis and broncho-pneumonia. *Gastric influenza* may commence suddenly with profuse vomiting and diarrhœa and accompanied by abdominal pain. In very young infants the disease may rapidly prove fatal, but in older children the symptoms subside

in 2 or 3 days. *Nervous symptoms* may complicate influenza, and the connexion between this disease and encephalitis is not yet fully explained. Influenzal meningitis is, however, well known and is described on p. 299.

Difficulties in diagnosis.—During an epidemic the diagnosis is extremely simple, especially when other members of the family are infected. The chief difficulty, however, arises in sporadic infections, and the name of " influenza " is often given without justification because the symptoms are so indefinite. Later the true nature of the patient's complaint is disclosed, and the mistake then becomes apparent. The diagnosis of influenza should not be made until every other possibility has been excluded, and then only with the very greatest misgiving. The blood-count will be found a great help, as influenza shares with typhoid fever the peculiarity that there is no leucocytosis but rather a tendency to leucopenia.

Prognosis.—On the whole the prognosis in children is excellent, the mortality being chiefly among those with pulmonary complications.

Treatment.—The child should, where possible, be kept apart from other members of the family, with a maximum supply of fresh air. The temperature of the room should be maintained at from 60° to 65°. Where catarrh of the upper respiratory tract is severe a cresolene vapour lamp may be used for one or two hours three times daily. Nasal douching with glyco-thymoline or liquid paraffin containing some antiseptic such as Chloretone Inhalant should be instituted. Half a tablet of aspirin (2½ grains) should be given three times daily to a child of five or six.

Diet.—Frequent drinks of sugar-water or fruit juice and water, well sweetened, should be given, the child being encouraged in this by keeping the drinks close at hand. When a desire for food has returned, starchy preparations, such as groats, Cream of Rice, or potato and gravy should be given at meal-times, the milk, which should be diluted, being limited to half or three quarters of a pint in the day. Once the tongue has

begun to clean up, a more solid and generous diet should be resumed at once. Where the temperature is high, tepid sponging with water at about 80° F. is useful.

Bowels.—The bowels should be kept well open. With infants this can be accomplished by giving milk of magnesia, a teaspoonful every hour until the bowels move. Where this is difficult, a saline enema of 3 or 4 ounces should be given repeatedly until a free action has taken place. In older children Syrup of Figs, a teaspoonful at bedtime, or grey powder, 3–5 grains, should be given. If there is continual difficulty with the bowels it is best to give one of the liquid paraffin preparations, such as Semprolin Emulsion or Petrolagar with phenolphthalein, one to two teaspoonfuls night and morning.

Complications.—Complications such as otitis media must be dealt with as described on p. 147. Where the symptoms are chiefly gastric, small infants should be given a dose of castor oil followed by 24 hours' starvation, and then very diluted feeds and a gradual return to normal. In older children, after the preliminary dose of castor oil, the child should be given sips of sugar-water or fruit juice and water, each with a pinch of bicarbonate of soda added. Full doses of bismuth subnitrate or carbonate, 20 grains four times daily, tend to "settle the stomach." The child should not return to milk until all signs of vomiting have ceased, and if the initial fluid is not retained saline with glucose must be given subcutaneously or intravenously.

It is the author's experience that some complication should be suspected if the temperature remains above 100° or 101° after the third day. A careful examination of the urine should be made in all cases.

Prophylaxis.—Against sporadic infections, a course of vaccines such as Public School Anti-Catarrh Vaccine (Parke, Davis & Co.) is undoubtedly of great value.

Vaccination

Vaccination remains the chief method of prophylaxis against smallpox, and children who have been vaccinated

are almost or entirely immune to the disease. It is best done in infancy, best of all in the first few months of life. It has been shown[1] that complications such as encephalitis only occur in older children who are being vaccinated for the first time. Complications and mortality from vaccination in infancy are nil.

Method.—The arm should be carefully cleansed with ether or ether soap and the calf lymph blown on at one or, at most, two spots. Then with a sterile needle or scalpel tiny scratches should be made just through the epidermis, but insufficient to draw blood. Immediately after this a sterile dressing should be applied and firmly strapped in place with adhesive plaster. In girls the leg, and preferably the outer surface of the calf, should be used for vaccination. There is a danger in choosing the region of the buttocks that the site of the vaccination may become infected. The following directions should be given to the mother:

1. The arm must be kept absolutely dry. If it should get wet in bathing, the dressing must be changed at once. Do not use boracic lint or powder.

2. Do not undo the pad or look at the arm for three days, provided it does not seem too tightly fastened or uncomfortable.

3. In three days commence undoing the dressing and looking for vesicles. Once they begin to appear the dressing must be changed every day. In this way the dressing is not allowed to stick to the blebs and no difficulty is experienced in changing it.

4. If the child is very restless it may be necessary to fasten the dressing with adhesive tape. A ring dressing of celluloid is very comfortable and convenient.

5. Once the vaccination "takes" it reaches its height by the seventh day and then commences to heal rapidly.

By the third day the vaccination should begin to "take," beginning as a red area. By the fifth or sixth day a vesicle is present. By the seventh day the maxi-

[1] "Post-Vaccinal Encephalitis," Report of the League of Nations Commission, *Lancet*, 1928, ii. 674.

mum inflammation has taken place, and from this point the whole thing subsides rapidly. When vaccination is carried out under aseptic conditions a minimum of reaction takes place and it can only be considered a trivial proceeding.

Post-vaccinal encephalitis.—Encephalitis is a rare complication of vaccination, and the nervous symptoms arise from 9 to 15 days after vaccination. It may start with drowsiness, vomiting and convulsions. Squint may be absent, trismus is often present. This complication occurs most commonly in older children, the infant and young child being missed, and it does not occur in those who have been previously vaccinated. After a short illness the child recovers completely, without sequelæ or after-effects. Since this is the only serious complication of vaccination, it would appear that it may be easily avoided by vaccination in infancy. The mortality from post-vaccinal encephalitis is moderately high.

The morbid anatomy somewhat resembles that of encephalitis lethargica (*see* p. 283).

Fever Diet

(In Pneumonia, Acute Infectious Fevers, Otitis Media, Mastoid Disease, etc.)

In acute febrile disturbances which are likely to run a short and fierce course, one of the most important considerations from the standpoint of patient, nurse and doctor is the diet. As a rule the child refuses food and wants drinks only. The tongue is dirty and coated, the breath offensive, and constipation is present, due both to confinement in bed and lack of solid diet on which the bowels can act. Along with every acute febrile disturbance acetone appears in the urine in large quantities. This is due to the body- and dietetic fat being incompletely metabolized because insufficient carbohydrate (sugar) is available. Fat is therefore an undesirable element in the diet of a person

with high fever, and despite recent work[1] where a concentrated form of the fat-soluble vitamins is strongly urged, the author considers that the cream of milk, butter, eggs and any other oily or fatty food should be excluded in these acute feverish conditions.

The constituents of the diet which throw least work on the body, and yet supply it with the calories it demands, in the most readily assimilable form, are starch and sugar. In the stomach starch breaks down into sugar. For these reasons fever diet should be along the following lines :

From one to two pints of plain water, sugar-water, barley-water or well-sweetened fruit juice and water, should be taken every 24 hours (2–4 years of age). It is well to keep a jug of such drinks within the child's sight as an invitation to him to take more. When the bowels have been well opened and the acetone has disappeared, in say 24 to 48 hours, despite the high temperature, the appetite will tend to return. If the child is able to take sufficient quantities at a time, the regular times of feeding, breakfast, dinner and tea, should be adhered to, but more frequent feeds may have to be attempted. At first starchy foods (*see* p. 67), made with water, should be administered, since they have a very high caloric value and can be made palatable. These can of course be made up with skimmed milk if desired. Certainly not more than half to three-quarters of a pint of milk, however, should be given in the day. Bovril is also useful. Broth made from veal, chicken or beef, with the fat removed, is to be recommended, preferably thickened with breadcrumbs or potato or other starchy substance.

To sum up, the diet in fever should contain a minimum of fat (butter, milk, cream and eggs) and a maximum of carbohydrate (sugars and starchy foods, such as cereals, bread and potato), with plenty of drinks and a very small quantity of protein. Glucose and barley-sugar are both advisable if vomiting is present.

[1] E. Mellanby and H. N. Green, "Vitamin A as an Anti-infective Agent: Its Use in the Treatment of Puerperal Septicæmia," *Brit. Med. Journ.*, No. 3569, June 1, 1929.

CHAPTER XVII

TUBERCULOSIS

General Remarks on Tuberculosis in Childhood

Broadly speaking, children are infected with the human and the bovine forms of the tubercle bacillus only, the human infection being very fatal and the bovine much less so.

Etiology.—Infection may take place:

(1) *By inhalation.*—This is undoubtedly the most common mode of introduction of the tubercle bacillus into the body. Contact with adults suffering from open tuberculosis can be proved in most fatal cases of tuberculosis in childhood. Infection from inhalation has its initial lesion most commonly in the lungs, often deep down near the pleural surface. A lesion may be extremely small and heal quickly, but may have infected the tracheo-bronchial glands before healing takes place.

(2) *By ingestion.*—The organism may be the human type, the child contaminating its fingers with dust containing the bacillus from the room and transferring the organisms to its mouth, or it may be the bovine variety ingested in milk. The latter mode of infection is common in certain parts of the country where the herds are infected with the tubercle bacillus. In this connexion it should be pointed out that the dangers of feeding a child with milk from a single cow are much greater than when mixed milk is used, as with the latter any tuberculous milk has been well diluted with that of healthy cows. The type of infection most often produced

by ingestion is abdominal tuberculosis (*see* p. 426). Milk can be completely sterilized by bringing it to the boil.

Various sites in the body.—In children probably the most common sites are the glands. Entrance is usually gained to the cervical glands through infected tonsils and to the mediastinal glands through a small infection of the lung itself, whereas in the abdominal glands it is gained through direct extension from the mucous membrane of the bowel. Very often the gland has been previously damaged before being infected with the tubercle bacillus; for instance, with septic teeth the glands draining this area become enlarged and indurated, and are very prone to become tuberculous, unless the teeth are attended to at once. This may also be true of tonsillar glands draining infected tonsils.

Tuberculosis of the alimentary tract is dealt with on p. 426. Here it is sufficient to say that the infection may be either human or bovine, most frequently the latter, derived from infected milk.

Skin tuberculosis is uncommon, but when it does occur the site may be almost any part of the body. Tuberculosis of the skin overlying the nose (lupus) is occasionally seen in children, but tuberculides of the neck, hands and upper portions of the body are less rare.

Tuberculosis of the lungs is considered elsewhere (p. 420). It is sufficient here to remark that it occurs as miliary tuberculosis or as consolidated areas, especially of the apex, as in the adult type. Both types are rapidly fatal.

Tuberculosis of the bones and joints suggests that there has been an infection of the bloodstream with the organism, which has been transported to the infected part by this route. In these cases infection is most commonly due to the bovine bacillus.

Tuberculosis of the kidneys is of extremely rare occurrence apart from miliary tuberculosis.

An infection of the brain and meninges can be taken as always secondary to some primary lesion elsewhere, and is as a rule a part of a miliary tuberculosis.

Prognosis.—After an infection of some part of the body a lesion may become healed and shut off by fibrous tissue. The immunity of the body is then raised against this infection. On the other hand the infection may extend to some adjacent part of the body, e.g. from the hilum glands to the actual lung itself, from the tonsil to the tonsillar glands, or from the mucous membrane of the bowel to the mesenteric glands, or the stomach and bowel may be indirectly infected from swallowed sputum.

The prognosis is always extremely grave when the bloodstream becomes infected. Tuberculosis confined to glands and joints is comparatively benign as opposed to an infection in some vital organ such as the brain. General miliary tuberculosis, which is the direct result of a blood infection, is always fatal, but fortunately the blood may become infected without distributing this infection so widely.

Diagnosis.—The clinical picture in all forms of tuberculosis should be given first place in diagnosis, and nothing should be allowed to overshadow it. As aids to diagnosis, however, X-rays are found of great help ; for example, calcified areas in the glands of neck, abdomen and chest are shown up in this way. Infected glands in the posterior mediastinum and small areas of calcification or active infection in the lungs also become evident with the aid of X-rays. Tuberculin tests must be used with the greatest care. It should be remembered that after the age of two an increasingly large proportion of children give a positive Pirquet test, and the author prefers the intracutaneous (Mantoux) test, as it is more easily controlled and more exact. A detailed account of the method will be found on p. 438. The giving of subcutaneous tuberculin cannot be considered to be without risk, as there is a focal, a local and a general reaction to an appropriate dose. The focal reaction may stir up a slumbering infection with disastrous results in childhood. Before a tuberculin test is done some definite focus of infection should be suspected. If the test is used

indiscriminately on children, when a positive reaction is obtained one is faced with the question whether the lesion is active or quiescent, and where it exists. The most satisfactory use of the intracutaneous tuberculin test, in the author's opinion, is to confirm a diagnosis, especially in very young children, of what appears clinically to be tuberculosis.

Treatment.—The best policy with tuberculosis is prevention, and this can only be carried out by isolating all tuberculous subjects and separating them from young children. The boiling of milk prevents ingestion of the disease. Once infection has taken place general treatment should be instituted at once. The child should live an open-air life and have plenty of sunshine, natural or artificial. The treatment of tuberculous lesions in childhood by tuberculin, apart from genito-urinary tuberculosis, is fraught with the gravest danger.

Pulmonary Tuberculosis

At the outset it must be made clear that, so far as our present knowledge goes, pulmonary tuberculosis in childhood, apart from miliary tuberculosis, is a comparative rarity.

Etiology and pathology.—The organism is inhaled and is almost invariably of the human type. A history of contact with an infected adult can nearly always be elicited. The initial lesion may be so small that it gives rise to few or no symptoms, and those which are present might be attributed to a mild influenzal attack. This tiny lesion, often deep in the lung, or near the pleural surface, may fibrose and heal, first, however, having infected the glands of the mediastinum draining that particular area. Ghon and Canti have shown that by the position of the infected tracheobronchial glands one can judge of the site of the original lesion, and by careful feeling of the crepitant lung with the fingers a small indurated area, the size of a pea, is often to be discovered.

If the child's resistance is lowered and the virulence of the organism great, the infected area does not show arrest of progress, but spreads rapidly and may involve a lobe. There may be areas of consolidation, which later caseate and break down, and the *adult type of tuberculosis* is then seen with cavity-formation. As a rule this picture is to be found in older children, and the lesion is in the upper lobe.

Mediastinal-gland tuberculosis, or hilum tuberculosis, results from the initial arrested infection, and its incidence in childhood is extremely difficult to ascertain, owing to the difficulty of diagnosing the disease.

Clinical picture.—A child with definite pulmonary tuberculosis of the adult type presents much the same clinical picture as the adult. Wasting, pyrexia, a constant short, hacking cough, with later a varying amount of sputum, and occasionally hæmoptysis, are the chief features. There is increasing dyspnœa and later actual cyanosis, if the involvement of the lung is extensive. Night sweats cannot be said to be typical of tuberculosis in childhood ; in fact they merely indicate that a child is not thriving properly. On examination the findings are those of consolidation, with or without cavity-formation, one lung only being involved in the early stages.

Where the lesion has healed and the mediastinal glands have become infected secondarily, a variety of symptoms are said to be present, such as lassitude, failure to thrive, pallor, anorexia, unexplained pyrexia. It is extremely difficult, however, to verify these facts. If the glands become active and matting takes place, and if they spread to the region of the hilum of the lung, certainly the symptoms just described will be present. The cough is of a peculiar character, brassy, dry and sometimes spasmodic. It reminds one at times of a foreign body in the bronchus, and at times of whooping-cough. On physical examination dullness in the region of the vertebral column and d'Espine's sign may be elicited, i.e. bronchial breathing is heard to the level of the fourth, fifth or sixth thoracic vertebra

instead of ceasing at the second. Neither of these signs, however, is diagnostic. Enlarged veins over the chest-wall and the presence of glands in the axillæ are signs of importance.

Diagnosis.—In the adult type of tuberculosis there is little or no difficulty in diagnosis. The organism is discovered in the sputum or in the gastric contents or fæces. The clinical picture, with continued pyrexia and wasting, coupled with the physical signs and X-ray appearances, should be diagnostic. The intracutaneous test gives additional confirmation. Fibroid lung or bronchiectasis is sometimes mistaken for pulmonary tuberculosis, but in the former the temperature is seldom continually elevated, cyanosis and clubbing are marked, and there is no history of contact with an infected adult. Moreover, the X-ray appearances are characteristic of bronchiectasis, and have not the fluffy, stippled appearance of tuberculosis. Lastly, the sputum is repeatedly negative when tested for the tubercle bacillus.

The diagnosis of mediastinal-gland tuberculosis, unless active and involving the hilum of the lung, presents the greatest difficulty. It is often suspected and antero-posterior and lateral X-rays are taken, but these give little or no conclusive evidence. Occasionally when the posterior mediastinum is encroached on by the enlarged glands and the cough is characteristic, a diagnosis can be made. The tuberculin tests are not helpful, as if positive they do not indicate the activity or otherwise of the lesion. On occasions it may be necessary to differentiate between whooping-cough and mediastinal tuberculosis, and sometimes it is only by observing the course of the disease that this is possible. A foreign body in the bronchus simulates closely the picture of tuberculosis of the mediastinal glands, and careful X-rays and often bronchoscopy may be necessary to differentiate between them.

Prognosis.—The adult type of pulmonary tuberculosis with gross involvement of the lungs is rapidly fatal.

In mediastinal tuberculosis there is a tendency to recovery. Post-mortem examinations have been performed on adults showing the presence of old caseous mediastinal glands so frequently that we now recognize this tendency. The glands may caseate and burst into a bronchus, disseminating the disease or involving a blood-vessel. The organisms may thus be scattered throughout the body, causing miliary tuberculosis. Mediastinal tuberculosis, therefore, remains a menace to the child. In a certain proportion of cases it tends to spread directly to the lungs and to involve these organs as a whole, with fatal results.

Treatment.—Once the diagnosis has been made, a maximum of fresh air and sunshine with a good generous diet should be given. The vitamins should be provided in the form of cod-liver oil, fresh fruit and vegetables. A diet high in milk, eggs, cream and butter is not recommended for children. In this type of tuberculosis natural or artificial sunshine should be given with the greatest caution and treatment by tuberculin is contra-indicated. For further details of treatment *see* pp. 420, 426 and 432.

Miliary Tuberculosis

Etiology and pathology.—Miliary tuberculosis as a primary lesion for practical purposes does not exist. In every case a primary focus will be found somewhere. In a series of 70 post-mortem examinations of cases of tuberculous meningitis (*see* p. 288), 87 per cent. showed a primary lesion in the gland at the bifurcation of the bronchi, and 13 per cent. in an infected abdominal gland. These infected and usually caseous glands tend to burst into some vessel near by, disseminating tubercle bacilli throughout the body via the blood-stream. The commonest site of this widespread infection is the meninges, giving rise to tuberculous meningitis, but concurrent with this and almost as common, is involvement of the lungs. In 75 per cent. of the cases the spleen, kidneys and liver were infected and

in 45 per cent. of the cases there were ulcers in the bowel.

Age-incidence.—The disease is most commonly found between the ages of one and three years (*see* Fig. 70).

Seasonal incidence.—The largest proportion of the cases are in the first quarter of the year, the explana-

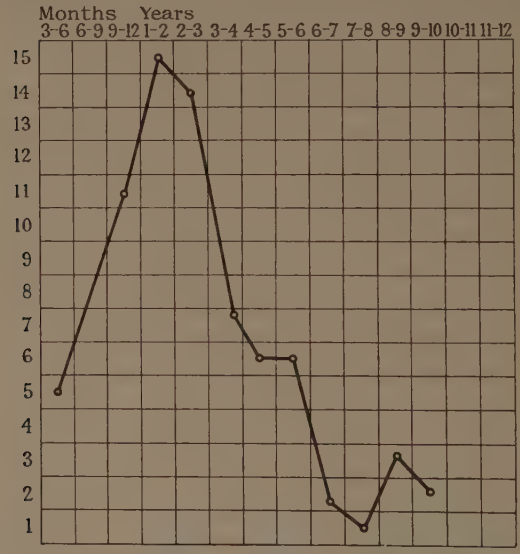

Fig. 70.—Age-incidence of miliary tuberculosis (tuberculous meningitis) in 70 post-mortem examinations. (*Practitioner.*)

tion being that at this time infectious diseases such as measles, whooping-cough and scarlet fever are rife. Moreover, in winter the weather is bad, confinement indoors is at its maximum, sunshine is scarce, fresh vegetables are difficult to obtain, and, cows being stall-fed at this time, milk is deficient in vitamins.

Clinical picture.—If the brunt of the infection has fallen on the lungs, the symptoms are largely

respiratory. The respirations are quickened, the child becomes cyanosed, the pulse is extremely rapid and he suffers from restlessness. There is wasting, slight cough, lack of appetite and extreme irritability. If the brunt of the infection falls rather on the meninges the symptoms are general lassitude, headache, drowsiness and irritability (*see* p. 289).

On examination probably the most significant feature of the case is the paucity of physical signs, although the child is obviously ill. There may be a few moist sounds scattered over the chest, but nothing definite. On palpation of the abdomen, especially when the chest is mainly involved, the spleen can often easily be felt. There is great significance in this fact. The temperature may be continually up, but in the early stages there are short bouts of pyrexia only. The rapidity of the pulse and respiration, with a tendency to cyanosis, is, however, a constant feature which should not be overlooked.

Diagnosis.—This is extremely difficult, especially early in the disease, because of the lack of physical signs. An X-ray of the chest shows typical fine, mottled or stippled appearances. On washing out the stomach or examining the stools of the child, tubercle bacilli may be discovered. With involvement of the meninges, meningitis is quickly suspected and proved. Very often typhoid or paratyphoid is suspected, but with a negative Widal test this can be ruled out. Examination of the urine excludes pyelitis, the general appearances of which may somewhat resemble miliary tuberculosis.

Prognosis and treatment.—In miliary tuberculosis all authentic cases are fatal. The treatment is therefore palliative. Oxygen relieves the dyspnœa and cyanosis, and with meningitis lumbar puncture should be used.

Prevention.—Since miliary tuberculosis is always secondary to a primary focus, there is no reason, once the primary focus has appeared, why the disease should not be arrested and its spread throughout the body

prevented. With the development of the primary lesion the child shows such symptoms as lassitude, wasting, lack of appetite, dry skin and hypotonia of the muscles. At this stage an investigation with X-rays and the intracutaneous tuberculin test (p. 438) may reveal the site of the primary focus. Now the general health should be raised; much fresh air, a change to the seaside, good food and sunlight are essential. After the age of six miliary tuberculosis is comparatively rare, and therefore the greatest care should be maintained until this age has been reached.

Abdominal Tuberculosis

It is proposed to deal with *tuberculous ulceration of the bowel*, *tabes mesenterica*, and both the ascitic and plastic forms of *tuberculous peritonitis* together, as it is common to find two or more of these conditions present at the same time. In fact they develop one from the other, and one expects to find all present to some extent in almost every case of abdominal tuberculosis.

Abdominal tuberculosis is usually due to a primary focus in the bowel, mesenteric glands or peritoneum, but sometimes, especially in the much older child, to a lesion secondary to some pulmonary focus.

Age-incidence.—The disease is most common during the second, third and fourth years, and uncommon after the age of 11. It is much more prevalent in the North of England and in Scotland than it is in the South of England, especially in London.

Etiology.—The geographical incidence of this disease is attributed by some to the difference in the milk supply, especially to the use of dried milks in England. The question arises whether abdominal tuberculosis is due to the bovine or to the human tubercle bacillus, and it is a very difficult question to answer, the authorities differing widely. Certainly children have developed abdominal tuberculosis who have had all their milk boiled. It is probable, however, that it is due to the human bacillus in a small number of cases, but to the bovine bacillus as a general rule.

The organism enters through the mucous membrane, spreads by way of the lymphatics to the mesenteric glands or to the peritoneum of the bowel, and thence to the rest of the peritoneal cavity. These three lesions, then, tabes mesenterica, peritonitis and ulceration of the bowel, are all so intimately bound up that they must be dealt with as one.

Pathology. **Tuberculous ulceration of the bowel.**—Probably the initial lesion is in the bowel submucosa. There a tiny tubercle is formed which ultimately bursts through the mucosa, leaving an ulcer with a narrow outlet into the bowel and a deep base with overhanging edges. This may become confluent with other ulcers arranged in a circular manner, so that when healing takes place the scar is circular and actual stenosis or occlusion of the bowel results. The infection may be so deeply placed in the submucosa that it extends actually through to the peritoneal surface, and perforation occasionally occurs, though this is, on the whole, uncommon. Tuberculous ulceration of the bowel is commonest in the small bowel for a few feet above the cæcum, and is not often found in the large gut. It may extend, however, throughout the whole length of the bowel and also in the colon. The ulcers may be present in great numbers.

Tabes mesenterica.—Passing back from the tubercle in the submucosa, organisms gain entrance to the lymphatic glands. The initial lesion in the mucosa may be too small to be recognized. The glands sometimes remain small and shotty, but at other times, depending on the health of the patient, attain or even exceed the size of an orange, caseate, liquefy and finally break down, discharging, as a rule, at the umbilicus.

Tuberculous peritonitis (*ascitic form*).—Tubercles may appear on the peritoneal surface, having spread from ulcers in the bowel, and these peritoneal tubercles spread rapidly from the bowel surface to the parietal peritoneum. A clear effusion forms in the abdominal cavity and the loops of bowel float about in this fluid. There may be very few adhesions between the bowel

loops at this stage. It is when healing commences, or after an operation when the effusion has been released, that the loops of bowel tend to adhere both to one another and to the parietal peritoneum. This is the so-called *plastic form of peritonitis*. The omentum may become grossly involved, the tubercles coalescing and actual caseous masses forming in it. The omentum tends to be drawn upward over the transverse colon, and a knobbly, doughy mass can be felt in the abdomen. In the plastic type there is a great tendency for loops of bowel to become adherent to the abdominal wall and for the bowel contents to burrow through and burst, draining at the navel—so-called fæcal fistula. Obstruction often occurs in this type, so that vomiting and abdominal pain are common. Perforation of an ulcer, resulting in septic peritonitis, is commonly the cause of death in these cases.

Symptoms and physical findings.—Probably the commonest complaint of a child with a tuberculous infection anywhere in the abdomen is pain. In *tuberculous ulceration of the bowel* the child may remain apparently perfectly fit physically, but in prolonged, severe cases rapid wasting occurs. The stools are loose and contain much mucus ; they are most offensive in odour and often light-coloured, as they are, for instance, in cœliac disease, to which there is some clinical resemblance. In slight cases the temperature is not raised, but in severe cases there is a swinging temperature varying between 101° and 99°.

In *tabes mesenterica* colicky pains in the abdomen, lassitude and failure to gain in weight are the common symptoms. On palpation little may be made out unless the child be asleep or under an anæsthetic, or will allow the abdominal wall to relax completely. When this is done the glands may be found. Sometimes there is a group situated at the root of the mesentery, in which case they are fixed ; at other times the glands are freely movable, as they lie in the mesentery itself. Unless the lesion is extensive there is no fever.

In the *ascitic form of tuberculous peritonitis* colicky

pains may have been present, directing attention to the abdomen, which is noted to be large and to contain fluid. It may be greatly distended and shiny, the vessels prominent over the surface. An examination may reveal dullness of the flanks, due to the fluid, and a mass of enlarged glands and rolled-up omentum may be felt on deep palpation. The child itself is wasted, anæmic and languid. There is no fever unless the infection is generalized.

In the *plastic form of tuberculous peritonitis* the abdomen feels doughy and the loops of bowel are bound one to the other. The whole abdomen is too firm, and, with relaxation of the abdominal wall, large caseous glands and the rolled-up omentum are easily made out. Very often when the lesion is great there is an irregular temperature, especially when the patient is not progressing well. There may be a tendency in both these forms of peritonitis for the umbilicus to pout, and especially is this the case in the plastic form, just prior to a fæcal fistula at the umbilicus.

Diagnosis.—Chronic diarrhœa in a child of over one year, especially if the stools contain mucus and at times shreds of blood, ought to suggest the possibility of **tuberculous ulceration of the intestine.** Cœliac disease, with chronic looseness of the bowels, large, pale, porridgy stools and a full lax abdomen, may cause some confusion in the diagnosis. The liver in cœliac disease, however, is not palpable, while it tends to enlarge in tuberculous ulceration. Too much milk, especially creamy milk and eggs, may cause chronic bowel looseness in older children. Here, however, the stools do not contain blood, and the removal of fat is quickly followed by a cessation of the symptoms. A careful examination of the stool by the antiformin method will often reveal the actual presence of the tubercle bacillus, and should be carried out wherever this diagnosis is seriously considered.

Tabes mesenterica.—For the purposes of diagnosis the child must be perfectly relaxed, and if necessary an anæsthetic should be given. Sufficient time should

be devoted to the diagnosis, the abdomen being carefully palpated in all parts, especially beneath the recti near the umbilicus. Where the masses are large, renal sarcoma must always be considered, in addition to fæcal masses. The presence of a large spleen in addition suggests the abdominal type of lymphadenoma, but this may not always be the case.

Tuberculous peritonitis (*ascitic form*).—The clinical picture is clear cut, an abdomen which is dull in its flanks and the child showing pallor and lassitude. Cardiac and renal ascites are excluded by examination of the heart and urine. There may be a differential diagnosis of cirrhosis of the liver, but in this condition the liver may be felt to be hard and knobbly, and there is a history of the condition having lasted for some time, with recurrent attacks of jaundice.

Plastic form.—This is to be distinguished from the encysted form of pneumococcal peritonitis, a condition which differs in that there is a history of fever at the commencement and extreme illness with a sudden onset. A generalized peritonitis due to appendicular abscess, in the event of a failure to recognize it, may also cause confusion, but the differentiation ought easily to be made by the history of the onset, which in peritonitis due to appendicular abscess is sudden. In the late stages, however, this differentiation is sometimes very difficult. There is also chronic intussusception where there has been a certain amount of generalized peritonitis. Here the omentum covering the mass of the intussusception gives the whole abdomen a lumpy, doughy feeling. In two such cases which the author has seen, a mistake was made in the diagnosis.

For further diagnostic tests *see* p. 422.

Treatment.—In treating most abdominal conditions, especially abdominal tuberculosis, rest in bed is essential. There is a natural tendency for the movements of the bowels to lessen and for constipation to occur while lying in a recumbent position, a condition which in abdominal tuberculosis is most desirable. In addition, the colicky pains diminish or disappear

altogether when the patient is in this position. The expenditure of energy is reduced and consequently less food is required, and thus less work is thrown on the stomach and bowel. Finally, the control of the child and its habits is made easier, so that the nursing becomes much simpler.

Apart from the damage to the body itself from absorbed toxins in *tuberculous ulceration of the bowel*, there is a much greater damage being done by malabsorption, due to the food being hurried through the bowel and imperfectly digested. This chronic intestinal indigestion must be treated by offering the bowel those portions of the foodstuff which are most easily digested and assimilated. A diet high in protein, low in fat and moderately high in carbohydrate is therefore found the most suitable, and since in *tabes mesenterica* and *tuberculous peritonitis* there is very often an accompanying ulceration of the bowel, such a diet should be adopted in these conditions also. Not only are the proteins the best-managed element of food, but they produce alkaline, constipated stools, which is an advantage. The diet should contain raw, scraped or underdone steak, a little well-skimmed boiled milk or junket made from skimmed milk, two or three eggs in the week minus most of the yolk, and crisply-done bacon from which most of the fat has been removed. The fat, being difficult to digest, is kept low, hence the skimmed milk and the egg without yolk. Carbohydrates are best given as starches, and then in a most digestible form. Bread should be given in the form of crisp toast or rusks, or it should be previously re-baked in the oven. The amount of potato should be limited to one small one in the day. Milk pudding should be cooked for three or four hours, the water being thrown away twice. It will be noticed that this diet is very much like that given for cœliac disease (*see* p. 115). As the child improves and the stools become normal a more liberal supply of fats (butter, milk, cream and eggs) should be given.

Sunshine.—This is a most valuable therapeutic

agent in treating tuberculous infections, especially abdominal tuberculosis. It must be administered in carefully graduated doses, starting with extremely little. The aim should be to get the abdomen, and in fact the whole child, sun-burnt. In patients who pigment, the prognosis is much better than in those who do not. Exposure to fresh moving air is also an advantage, and on this account sea air is better than inland air, other things being equal. When the weather permits the clothes should be reduced to a minimum and the limbs exposed to the direct sunlight and fresh air during part of the day.

Probably no therapeutic measure is more important in this disease than the administration of cod-liver oil. Its effect appears to be to raise the immunity of the child to the tubercle bacillus and to induce healing, particularly when the oil is well borne. This is especially important, as it has been noted that a high-fat diet is not well borne, and thus the vitamins must be administered in as concentrated a form as possible.

Prognosis.—In Scotland, where abdominal tuberculosis has for many years been extremely common, there is a saying that the children under two die and those over two live. Broadly speaking this is undoubtedly true. Certainly the younger the child the more the tubercles tend to become generalized. The mortality becomes very small over the age of three. In *tabes mesenterica*, where the glands caseate, liquefy and drain at the umbilicus, or where a fæcal fistula has developed, the outlook, though grave, is not considered hopeless, and a large proportion of the cases recover. If the *ulceration of the bowel* is extreme there is a tendency to stenosis afterwards, with chronic ill-health and intermittent obstruction. In *tuberculous peritonitis*, unless the health of the child is extremely low and the infection becomes generalized, the outlook is excellent. The ascitic form answers readily to simple drainage, and the plastic form clears up entirely with the general treatment outlined above.

Tuberculous Cervical Adenitis

Glandular tuberculosis is the commonest lesion in childhood. Infection of the abdominal glands has been dealt with elsewhere (p. 426), as also has infection of the mediastinal glands (p. 421).

Etiology and pathology.—The cervical glands most commonly infected are those which lie at the angle of the jaw and serve to drain the tonsil. As a rule there have been one or two attacks of sore throat, with the result that the tonsillar glands have become permanently enlarged, as also have some of the other cervical glands. There may be an interval of some weeks or months when there is a constant increase in the size of the glands on one or both sides. This is due to an infection with the tubercle bacillus which has gained entrance through the damaged tonsil and has reached the already affected gland. Inflammation occurs and may rapidly pass on to a cold abscess, or, healing, may be followed by caseation. The infection may not always manifest itself in the tonsillar gland. In some cases the glands deep to the sternomastoid or posterior to it may be the first to be involved; usually several become enlarged.

Clinical picture.—The child with slightly enlarged cervical glands may be discovered to have a large swelling half the size of a tangerine orange over the side of the neck. This swelling may appear slowly, or in the course of a few hours, and there may or may not be a temperature, usually there is not. On slight pressure over the swelling no great tenderness is complained of, and at first, if the swelling is deep-seated, no fluctuation is noted. Later, as the pus nears the surface, the skin becomes red and shiny and distinct fluctuation is noticed. If no treatment is instituted the skin breaks down and a sinus is formed.

Treatment.—Cervical adenitis should always suggest the possibility of tuberculosis.

Removal of septic foci.—The first step is the removal of septic teeth and tonsils. It is usually

through the tonsils that the glands become infected. If there is no considerable enlargement of the other glands apart from the tonsillar glands an intracutaneous tuberculin test is a help in the diagnosis.

Conservative treatment.—Splinting of the neck with a back-splint and foot-piece limits the movements of the head in all directions (Fig. 71). When head movement is prevented the cervical glands are not pressed upon by the neck muscles and thus obtain complete rest. Should the glands not react favourably to conservative treatment and there is a tendency to matting of the glands and abscess-formation, surgical aid should be called in before this process has been allowed to go too far. The work of the surgeon is made much more difficult if he must deal with the situation when inflammation is well advanced.

Fig. 71.—Method of applying an ordinary foot splint to the head for cervical adenitis.

Sunshine and ultra-violet rays.—Exposure of the affected area of the body to sunshine or ultra-violet rays is strongly to be recommended if carried out with care. Just as in pulmonary tuberculosis, this powerful therapeutic agent must be used with caution.

Tuberculin injections, except as adjuncts to diagnosis, should not be employed.

General treatment.—Attempts should be made to

improve the patient's general health by a change to the seaside, a well-balanced diet, a maximum of fresh air and rest, with some cod-liver oil preparation if this be well borne.

Pleurisy

Pleurisy with effusion may be divided into two types —purulent and non-purulent. The purulent type has already been described under the heading of empyema (*see* p. 210). The non-purulent type is for practical purposes always tuberculous. It must be remembered, however, that there is a stage in every effusion when it appears to be quite clear.

TUBERCULOUS PLEURISY

Pathology.—The pathology of this condition is well understood. The pleural surface of the lung is studded with small tubercles, which tend to "weep" into the pleural cavity. The fluid, which may collect slowly or rapidly, is pale, straw-coloured and clear or almost clear. Lymphocytes may, however, be present in moderate quantities. The tubercles may spread throughout the whole pleural surface, causing a thickened, red, shaggy appearance, with a tendency to the formation of adhesions between the two layers of the pleuræ.

Symptoms and clinical picture.—An attack may commence with sharp pain in the side, accompanied by a high temperature lasting for two or three days or even a week. The pain, especially on inspiration, quickly passes off. On examination friction may be heard, but more likely an effusion, which may be large or small, is discovered.

Prognosis and treatment.—Pleurisy is comparatively uncommon in childhood, unless associated with the adult type of pulmonary tuberculosis. X-ray examination in such cases will often reveal some primary focus in the glands or lungs, from which the lesion has spread. Unless the primary lesion in the lung is

too gross the ultimate outlook is excellent. In most cases the pleural surfaces heal and the whole condition subsides.

As in other forms of tuberculosis, the first step is to put the lung as far as possible at rest. This is often best done by strapping the chest in a direction across the ribs, using broad bands of adhesive tape or plaster, one strap overlapping another until the whole chest-wall is splinted. This, by preventing the lung on the affected side expanding or contracting to any great extent, prevents the pleural surfaces from rubbing against each other. It may be necessary, where a large effusion is present and the heart is much displaced, to insert a needle and withdraw the fluid, but on the whole it is preferable, provided the diagnosis admits of no doubt, to refrain from tapping the chest. There is always a risk of carrying into an already infected pleural cavity other foreign organisms with disastrous results. Repeated X-ray examinations are of very great value in assessing the progress of the patient, as they show not only the presence or absence of the effusion, but also the progress of the underlying pulmonary condition.

Treatment directed towards the improvement of the general health of the patient has been described under other tuberculous infections (*see* pp. 425, 432).

Bone Tuberculosis

This subject is dealt with more comprehensively in textbooks on surgical tuberculosis[1] than is possible in the present volume, where space permits of only a broad survey of the subject.

Etiology and pathology.—The infection gains entrance to the bones by way of the bloodstream. As a rule there is some primary focus elsewhere in the body, and bone tuberculosis is usually secondary to glandular tuberculosis in the mesentery or mediastinum. Undoubtedly the bovine infection is much more

[1] E.g., John Fraser, "The Surgery of Childhood."

frequently responsible than the human type, and bone tuberculosis is actually more prevalent in districts where the milk supply is infected.

Clinical picture.—The sites of the various lesions in bone tuberculosis should be mentioned. *Tuberculous dactylitis* is not uncommon and is especially found in the proximal and middle phalanges, particularly in infants and very young children. Tuberculosis of the *hips, knees and ankles* is to be found in this order of frequency.

A *tuberculous hip* is suggested by chronic pain in the hip, especially if it wakes the child at night. Very often X-rays when employed in the early stages are of no help and may even be misleading. The clinical picture of a child with a slightly flexed hip, with a certain degree of fixation due to spasm of the muscles, and often showing a temperature, is in itself classic. It should certainly be relied on rather than a negative X-ray examination, especially in the early stages. The intracutaneous tuberculin test is of great help in doubtful cases.

Caries of the spine (*see* Fig. 61, p. 364) is fortunately becoming more rare. It may occur at any age, but is more common after than before the age of 2 years. The disease may be found in the cervical, dorsal or lumbar spine, the mid-dorsal region being probably the most common site. Among the early symptoms is that of wasting, and later there is pain on movement, with deformity of the spine due to collapse or fixation of some of the bodies of the vertebræ.

Treatment.—The primary consideration is, as in other inflammatory conditions, to put the diseased part at rest. *Spinal cases* should be kept flat on a board or in a plaster cast or jacket, depending on the site and severity of the lesion. *Hips, knees and ankles* should be given complete rest in a plaster cast or with a splint. *Fingers* also require splinting.

The general treatment for tuberculosis described on pp. 425, 432 should be instituted, and every effort made to raise the child's immunity.

The Intracutaneous Tuberculin (Mantoux) Test

Method of Making up Solution and Technique of Test

It is best to use a tuberculin syringe. The amount of diluted tuberculin and control fluid is in each case one tenth (0.1) c.c., and to measure this volume satisfactorily such a syringe is necessary. After some practice, however, the amount can be roughly guessed by the size of the little whitish lump which the intradermal injection of 0.1 c.c. produces. Two syringes are necessary, one for the tuberculin, the other for the control fluid, and if they are clearly marked, there will be no risk of contamination of the control syringe by tuberculin. The ordinary hypodermic needle can be used, however, provided the point is not too long and tapering.

Use Koch's Old Tuberculin. Bovine tuberculin is not necessary, as a positive reaction in the case of bovine infection, e.g. bone and abdominal tuberculosis, will be given just as readily to a test with human as with bovine tuberculin.

Tuberculin dilutions:

Solution No. 1 consists of 1 mgm. in 0.1 c.c., a dilution of 1 in 100. For this add 0.5 c.c. of Old Tuberculin to normal saline, making up to 50 c.c. Inject 0.1 c.c.

Solution No. 2 consists of 0.1 mgm. in 0.1 c.c., a dilution of 1 in 1000. For this take 1 c.c. of solution No. 1 and add to 9 c.c. of normal saline. Inject 0.1 c.c.

Control solutions should be made up in the same way, using glycerin broth instead of tuberculin. The normal saline used should be sterilized and have 0.25 c.c. of pure carbolic added to each 100 c.c. of the saline.

For the test, use Solution No. 2 in most cases, that is one-tenth mgm. Repeat the test if necessary with Solution No. 1. When one-tenth mgm. is being used, it must be remembered to use the same dilution of tuberculin and of the control of glycerin broth. The solutions should be made freshly about every two weeks and kept in an ice-chest when not in use. The

results should be read in 24 and 40 hours. In very scrofulous children, or those with phlyctenular conjunctivitis, in whom a marked positive reaction may be expected, it may be wise to test first with one-tenth of a mgm. For this dilute Solution No. 2, one part, with nine parts of carbolized saline and inject one-tenth of a c.c. The solutions when prepared should be kept in sterilized brown vaccine or insulin bottles.

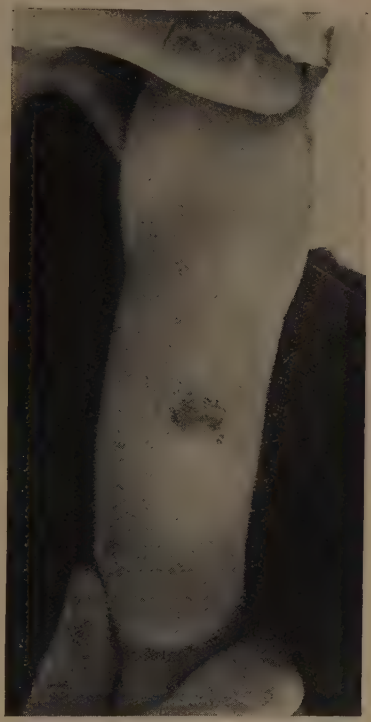

Fig. 72.—Positive intracutaneous tuberculin (Mantoux) test.

Cases where the test is applicable.—It is best not to do this test except where some lesion in a gland, chest or bone requires explanation. The promiscuous use of the test will often cause great embarrassment, especially in older children, as more and more difficulty will be experienced in finding the lesion. The test is of most use in infancy and early childhood, and ceases to be of great value after the age of four or five. The following directions are used by the author after the test has been performed.

Of the two marks on the child's arm the upper one is the control and the lower one is the intracutaneous tuberculin test. If the test is positive, the lower mark

will come up in from 24 to 48 hours as a red, indurated area the size of a sixpence and last two or three weeks (Fig. 72). If it is negative, both marks will die away. A positive test suggests:

(*a*) That the child has an active tuberculous lesion, or

(*b*) That the lesion is an old one, now healed.

If the test is positive and the site of the lesion is not evident, an X-ray of the child's chest is advisable, both antero-posterior and lateral views. If the test is negative, it is not necessary to do anything, as this excludes the possibility of tuberculosis in the child.

CHAPTER XVIII

RHEUMATISM

The rheumatic state includes a variety of manifestations of rheumatism affecting various organs and differing widely in the clinical picture to which it gives rise. Since we have no certain test or criterion to rely upon, the diagnosis is extremely difficult and therefore is very often wrongly made. With the exception of heart disease, nodules and chorea, most of the other manifestations cannot by themselves be taken as definitely rheumatic.

Etiology. Organisms.—For 25 years a controversy has raged as to the etiology of rheumatism. Evidence attributing it to an infection was first put forward by Poynton and Payne, who claimed to have made the discovery of a diplococcus in the heart's blood, in nodules, in damaged valves and, later, in cases of chorea, in the brain. Various workers have entirely failed to substantiate this, while some have, in part, borne it out. It must, however, be admitted that up to the moment it is by far the most attractive theory. There can be little doubt that, whatever the organism be, this disease is due to an infection, and everything points to the infection gaining entrance through the tonsils. Once the infection has taken place it cannot be said to have been eliminated, although many years may have elapsed from the onset.

Sex-incidence.—Statistics show the disease to be rather more common in females than in males, but with a great difference in the manifestations in the two sexes. Chorea is more common in girls than in boys, whereas arthritis is more common in boys than

in girls. Heart disease is equally common in the two sexes.

Heredity.—A history of an hereditary tendency can be elicited in about half the cases.

Age.—The age at which rheumatism starts is about three years, after which it increases very slowly to the age of five. At five the increase is extremely rapid, reaching its maximum about seven years of age, and from thence to twelve years there is a steady fall. In both girls and boys, therefore, seven is the age at which rheumatism is most prevalent (Fig. 73).

Fig. 73.—Age-incidence of initial attack in 172 cases of rheumatism.

Environment.—Repeated investigations have shown that there is a very close connexion between dampness and rheumatism. Clay soil, which prevents water draining away properly, dwellings near rivers and insanitary houses are all, in some way, associated with this disease.

Seasonal variation.—In an analysis of 172 cases in the year 1919-1920[1] it was noted that the majority of the attacks occurred in November and April, and that the weather in those two months was extremely inclement, being both cold and wet. Rheumatism is

[1] F. J. Poynton, Donald Paterson and J. C. Spence, "Acute Rheumatism in Children," *Lancet*, 1920, ii. 1086.

associated rather with the kind of weather than with any particular month in the year.

Scarlet fever.—Rheumatic carditis may first make its appearance shortly after a severe infection with scarlet fever. This fact can be ascertained in a moderate proportion of the cases and is suggestive as to the etiology.

Strata of society affected by rheumatism.—It is interesting to note the class of person which rheumatism attacks. It is not the well-to-do, in fact it is most uncommon in those who are better off financially. Nor is it prevalent among the labouring classes. Rheumatism is a disease of the intellectual poor—the clerk who is ambitious and clever, and inadequately fed and nourished, is the typical victim. Devoting more of his money to appearance and education than to food, he forms the ideal soil for the disease, while the labourer who makes no attempt to keep up appearance, but feeds himself comparatively well, appears to be more or less immune. The fact that it is people with the ambitious outlook of mind who are affected necessitates treatment accordingly. They are unwilling, or too keen on their work, to convalesce for the proper period. Gradually, however, the attitude of the public is being influenced by education, and it is now less common to find the treatment of rheumatism neglected.

Geographical incidence.—Rheumatism is extremely prevalent in the British Isles, while in some Continental cities it is practically unknown. In America the incidence is probably nowhere so high as in and about London.

Pathology.—The organs attacked by rheumatic infection most rapidly are the heart, its coverings and its valves, the subcutaneous tissue, especially round about the joints, the synovial membrane of the joints themselves, the brain and the tonsils. The most characteristic lesions of rheumatism are to be found in the heart's muscle and its valves and in subcutaneous nodules. These consist in a central fibrinous area,

which stains badly and shows no particular structure, and an area surrounding this containing endothelial cells intermixed with fibroblasts and inflammatory lymphocytes. In the heart-muscle itself such nodules are accompanied by large inflammatory cells containing many nuclei. Such an exudate and inflammatory reaction with multinucleated cells present is known as an *Aschoff's nodule*. The inflammatory reaction in a valve itself develops with the duration of the lesion. In an early lesion the valve is swollen and there is tendency to poor staining, with the presence of lymphocytes and inflammatory cells. Later, a fibrinous structureless exudate seems to extrude itself on the free surface of the valve. This stains badly, and on the free surface of the exudate the various circulating blood elements tend to deposit themselves. Deep to the fibrinous material, however, endothelial cells, lymphocytes and early fibroblasts appear, so that the essential structure of a damaged valve is identical with that of the subcutaneous nodule or heart-nodule previously mentioned. It is seldom that in rheumatism one portion of the heart alone is affected, that is, rheumatic endocarditis is seldom if ever present without an affection of the myocardium. Certainly rheumatic pericarditis does not exist apart from myocardial and endocardial changes. The term *rheumatic carditis* is therefore much to be preferred. It would seem that a tendency to a rheumatic " fibrosis " which is extremely widespread must infallibly produce permanent damage to the organs involved.

Chorea (Rheumatism of the Nervous System, Rheumatic Encephalitis)

Etiology.—Chorea or St. Vitus's Dance has been thought by some to be due to fright, by others to shocks or falls, but the more generally accepted theory is that it is a rheumatic encephalitis. A close study of the clinical picture and pathology supports this theory. Air raids were said to have produced much chorea

during the War, but an analysis of some 84 cases just after the War, and also of those cases occurring throughout the war months at the Hospital for Sick Children, Great Ormond Street, did not in the least bear this out. Chorea was shown to be seasonal, before, during and after the War, and therefore, if the older theory is to remain tenable, fright would have to be seasonal also.

Sex.—Authorities differ as to whether chorea is twice or three times as common in females as in males, but the preponderance of females is undoubted. No really adequate explanation of this disproportion is offered.

Pathology.—The morbidity is so low that very little material has been examined post mortem. Those cases carefully investigated, however, have shown a characteristic cellular infiltration around about the small capillaries, comparable up to a point with the picture of encephalitis lethargica. The whole of the brain is affected, especially the white matter, and in one case the caudate nucleus had suffered grossly; minute emboli and blocking of the vessels by thrombi were also noted.[1] In some cases extravasation of blood from the finest capillaries has been noted.

Clinical picture.—Usually chorea occurs at about the school age, that is between five and seven. The child is often of the bright, slim, rather nervous type. Some authorities claim that those with auburn hair are more prone to this infection. The child may be noticed to flag easily, to become unduly emotional, laughing or crying too easily, and to have lost colour. The tendency to drop or spill things is common, the writing becomes less legible, the speech less articulate. The movements in standing up or sitting down in a chair are impetuous. The child tends to fall about, catching one foot behind the other, and on this account is often covered with bruises or abrasions, especially about the knees. As the disease develops, all the muscles in the body become involved, even the eye-muscles. The involvement of

[1] J. G. Greenfield and J. M. Wolfsohn, "Pathology of Sydenham's Chorea," *Lancet*, Sept. 16, 1922, pp. 603–606.

the facial muscles causes grimaces, so that the child appears to smile or frown for no reason whatever. Constant shrugging movements of the shoulders and involuntary movements of the arms, hands, legs and feet are found. The respiratory muscles are attacked also, resulting in inco-ordination between the diaphragm and intercostals, most noticeable when the physician examines the child's chest. On these occasions, halfway through an inspiration or expiration, there is a sudden antagonism between the two sets of muscles, so that respiration may come to a standstill midway. The knee-jerks are active, giving rise to what is known as the " hung up " knee-jerk, that is, after the jerk has occurred the leg fails to drop back into its previous position for a moment. Snorting noises and clucking with the tongue are also characteristic. These constant movements continue throughout the child's waking period and tend to keep her from sleep. She becomes worn out and thin, and it is with difficulty that she can be fed. Bursts of crying further depress her. A pronounced case of chorea is a pitiful sight, and in some of the more severe cases the child may become almost maniacal. Accompanying most cases there is at least cardiac dilatation, but actual carditis frequently develops. The majority of cases sooner or later develop a heart lesion, in fact by the second or third attack at least 75 per cent. are so affected. The disease is also commonly accompanied by rheumatic nodules, but arthritis does not coincide with the chorea. As many as eight or nine attacks have been known to occur.

Prognosis.—From the chorea itself there is very little to be feared, that is, there is no evidence of any permanent cerebral damage, recovery appearing to be complete. It is in the accompanying heart disease that the danger lies. This aspect will be dealt with later.

Treatment.—There can be little doubt that a child with any rheumatic affection, especially chorea, recovers more quickly when taken out of the environment in which the infection occurred. A dry, warm house and climate are most to be desired.

General management.—Every patient with chorea ought to be confined to bed during the active stages. Choreiform movements mean that the rheumatic infection is present and is active, and the heart is therefore in danger. The child should be placed flat in bed with either a very thin pillow or none at all. Restraint in the form of shoulder-straps should be provided if there is danger of the patient sitting up. The sides of the bed should be padded in order to prevent banging of the head and limbs. The taking of the pulse is much more important than the temperature, in that it furnishes an indication of the cardiac condition.

Nursing.—Much skill is required in the management of chorea patients. They must very often be fed from a feeding-cup, and in the more severe cases should never be allowed to feed themselves. They require great tact in their management, as with their emotions so unstable any attempt at discipline must be discontinued.

Diet.—The maximum amount of food which they are able to assimilate without overloading the intestinal tract should be given, but it is advisable to confine feeding to three meals per day as far as possible. Not much fatty food should be given, and a diet rich in milk, cream and eggs is to be avoided. For a specimen Diet Sheet *see* p. 134.

Drugs.—A great variety of drugs has been employed in the treatment of chorea. *Aspirin* or *salicylate of soda* may be given during the acute stages—this may be done rather on general lines, as it does not appear to have any specific effect on the chorea. Aspirin, 3 grains three times daily, may be continued safely for some weeks. Sedative drugs are best used in the chronic stages. *Arsenic* in the form of *Fowler's solution* has been recommended, one minim three times daily, increased daily until signs of arsenic overdose have been reached, when the drug is discontinued. No good effects have accrued from such treatment in the author's hands. To understand the drug treatment of chorea it must be realized that there is an organic

and a functional element. During the acute stages the disease is almost entirely organic. In six or eight weeks, however, provided the pulse has settled and the condition of the heart is satisfactory, it may be safely assumed that the movements are largely functional, and treatment should therefore be instituted along these lines. It will be found that in such cases treatment with sedative drugs meets with rapid success, and it is probably in this stage of functional derangement that such drugs as *trional* and *chloretone* have gained their reputation in chorea. The mode of administration is to give 1 grain of either of these preparations four-hourly, increasing the dose steadily until 6 or even 7 grains are being given four-hourly. On such a dose the child becomes extremely drowsy. The drug should then be stopped entirely, and as it works off the movements will have ceased. *Luminal sodium* ($\frac{1}{2}$ grain), together with a little bromide (4 grains), made up to 2 drachms with chloroform-water and given night and morning, will act in a similar fashion during this stage.

Re-education.—During the chronic stage the functional element is greatly improved by attempts at re-educating the muscles. If the patient is given such occupations as knitting and sewing, walking along a mark on the floor or feeding herself, improvement of the finer movements of co-ordination is encouraged.

Removal of tonsils.—This measure will be dealt with more fully elsewhere, but it should be said that in all cases where the tonsils are enlarged and septic they ought to be removed.

Other Rheumatic Manifestations

In addition to chorea, rheumatism is liable to attack the *heart* (carditis), the *joints* (arthritis), the *muscles* (myositis), the *subcutaneous tissues* (nodules), the *throat* (tonsillitis) and the *skin* (rheumatic erythema and purpura) (Fig. 74).

Clinical picture.—The general appearance to which advancing rheumatism gives rise is the same whatever the system attacked. The child flags, becomes

pale and listless, loses weight and appears definitely anæmic. There is a complaint of pains in the limbs,

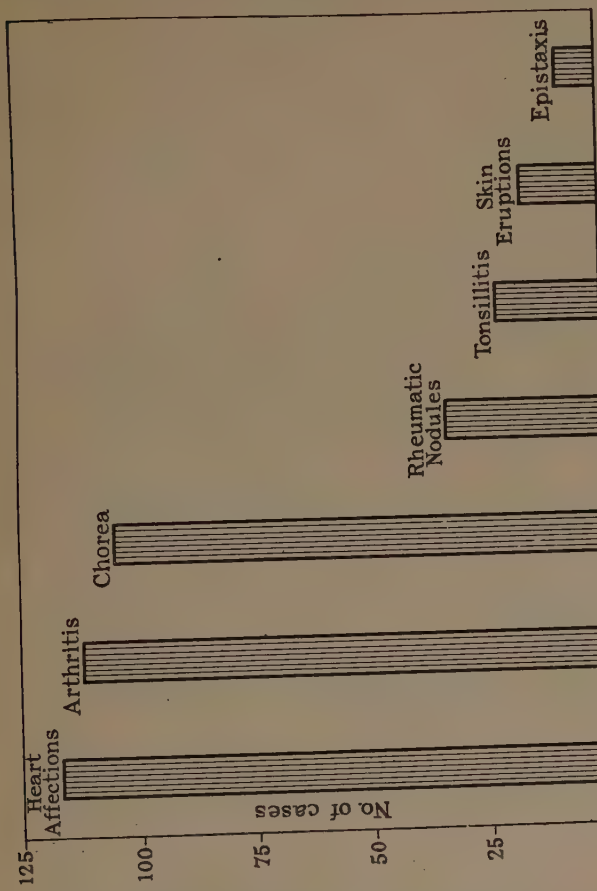

Fig. 74.—Table showing the relative incidence of symptoms in 172 cases of rheumatism in childhood. (*Lancet.*)

down the thighs, in the bones themselves and in the shins. These are termed "growing pains." The pains

are often also present in the shoulders and arms, in the back, and in the spine at the back of the neck. Stiff neck and cricks in the neck are common, and some children may get a persistent stitch in the side, which, it is suggested, is really an early muscular infection. As a rule there are recurrent attacks of sore throat, with enlargement of the tonsillar glands, though the enlargement may not be noticeable. There may have been a sharp attack of scarlet fever, or the child may have complained of breathlessness and palpitation after exertion. Such symptoms as those described are seldom found in the well-nourished; almost invariably the children are much under weight. A dirty tongue is a constant accompaniment of active rheumatism.

Nodules.—These are to be looked for over the patellæ, the elbows and the back of the skull, also over the tips of the spines of the vertebræ, on the knuckles and the ankles and on the spines of the scapulæ. On bending the knee, elbow, fist or ankle slightly so as to draw the skin tensely over the bony prominences, the nodules stand out beneath the skin as white marks (Fig. 75). Pressed on slightly, they slip and slide beneath the finger. An excision of a nodule shows it to be a localized inflammatory focus. Nodules may appear extremely rapidly and disappear just as rapidly; even within a day a well-marked nodule may be gone. They are a sign of extremely active rheumatic infection and are generally accompanied by a gross cardiac lesion. They have been noted, however, where very little carditis appears to be present. Of 33 cases with nodules noted by the author, 37 per cent. were fatal and 47 per cent. had very severe heart disease. In only 2 cases was the heart free.

Arthritis.—Unlike the picture in adults, very little is seen on examination of the affected joints in children. There is seldom any swelling, redness or heat, although the pain complained of may be excruciating and the child may resist the slightest movement. Occasionally, however, the adult type with swelling

is found. In the more severe infections accompanying arthritis there is involvement of the vertebral articulations. This is noted in the neck and back. In such a case the patient is unable to move the head in any direction and a mistaken diagnosis of meningitis has been made. Arthritis is much commoner in boys than in girls, possibly two boys to one girl, and in this way the disproportion in the sexes seen in chorea is equalized. As a rule an attack of tonsillitis precedes or is coincident with an attack of arthritis.

Tonsillitis.—So often does tonsillitis accompany or

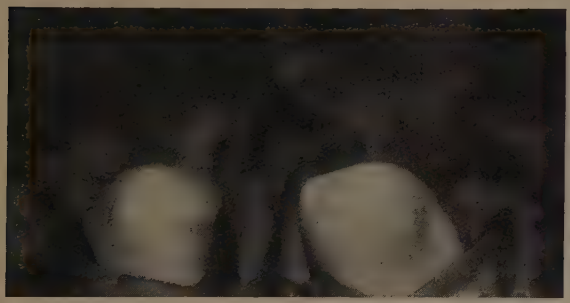

Fig. 75.—Rheumatic nodules on knees

precede rheumatism that it is reasonable to believe that the tonsils are the portal through which the infection enters the body. The appearance of an acute rheumatic tonsillitis is characteristic, there being a thin, pale exudate on the tonsil, as if the child had been recently drinking skimmed milk.

The skin.—Two types of rash are to be noted: (1) *rheumatic erythema*, which appears over the chest and back, occupying a great area; the edge is characteristically serpiginous or map-like, changing from hour to hour; (2) *rheumatic purpura*, which is seldom seen except in those malignant or fatal cases where the cardiac vegetations are luxuriant. As a rule these cases are fatal (Plate 10).

RHEUMATIC CARDITIS

The whole significance of rheumatism depends upon the extent to which the heart is implicated. On its involvement very largely depends the diagnosis in the majority of cases. Vague limb-pains without cardiac involvement may or may not mean rheumatism; with cardiac involvement the diagnosis is confirmed. When the heart is involved to a minor degree it is merely dilated. This is often seen early in cases of chorea and arthritis. On examination the apex-beat is found to be slightly outside the nipple-line, and on auscultation at the apex there is a very soft systolic murmur, probably due to regurgitation at the mitral valve from a dilatation of the mitral ring. With a more severe and prolonged infection, in addition to dilatation the valves are involved, the mitral valve being by far the most commonly affected, with production of regurgitation. During the healing of severe mitral involvement, stenosis of this valve almost invariably results. Mitral stenosis and regurgitation is the end-result of a vast proportion of rheumatic cardiac infections. In *mitral regurgitation* the heart on examination is found to be enlarged to the left, due to an hypertrophy of the left auricle and to a lesser degree of the left ventricle. No thrill is present over the præcordia. On auscultation a murmur, systolic in time, and propagated into the axilla, is noted. This murmur can also be traced backward to the level of or beyond the nipple-line. Where *mitral stenosis* and regurgitation co-exist there is also enlargement of the heart to the left, but in addition there is a distinct thrill over the præcordia. If the heart is acting well the first sound is obliterated by the presystolic and the systolic murmur, and the second sound is short and sharp. Where the heart works less perfectly and compensation is not so good, the presystolic murmur breaks off and moves forward so that it becomes mid-diastolic. This may be imitated by the sounds "too-ti-froo." When compensation and function are still less perfect this presystolic murmur may become

actually diastolic. In this case it is imitated by the sounds made by bellows and is known as a "bellows murmur." The blast outward of the bellows represents the systolic and the indrawing of air the diastolic murmur.

The *aortic valve* is the next most commonly attacked. Regurgitation results, but as a rule this does not occur until after the mitral involvement. On examination in the aortic area of the child, which lies to the left and not to the right of the sternum as in adults, the second sound is replaced by a soft murmur, the heart as a rule showing hypertrophy of the left ventricle. Aortic stenosis is still less common.

Tricuspid involvement is extremely rare and pulmonary involvement practically unknown.

Where there is an active rheumatic infection in the heart the *pulse is almost always increased in rate*, this being so universal that the pulse-rate is of extreme importance and ought to be firmly relied upon. The charting of pulse-rates is much more helpful than the taking of the temperature.

Rheumatic pericarditis.—Along with acute tonsillitis, rheumatic pericarditis is remarkable in that it gives rise to a high temperature. Most other rheumatic infections are not accompanied by much fever, especially if salicylates are being given. Despite salicylate therapy, however, pericarditis runs a high fever for some days or even weeks, the child appearing greatly distressed and having a pinched appearance. There is dyspnœa and often a short hacking cough reminiscent of pneumonia. On examination of the chest there is often a scattered bronchitis, but at the left base in almost every case, at the angle of the scapula, will be found a patch of high-pitched tubular breathing. The cause of this is debatable, some regarding it as a rheumatic pneumonia and others attributing it to compression of the left bronchus by the enlarged heart and a consequent collapse. The heart is enlarged and the pulse rapid. On auscultation a to-and-fro murmur, harsh in character, most audible over the sternum, is

heard. Seldom is there much fluid present, consequently the heart-sounds are not damped down or distant. Actually, if examined post mortem both the parietal and visceral surfaces of the pericardial sac are covered with a thick, felted coating which in many places is adherent, with possibly a very small quantity of opalescent fluid lying in loculated areas.

As in other rheumatic infections, correct nursing is essential, the child being kept very flat and still. The application of an ice-bag is helpful in many cases of præcordial pain. Digitalis therapy should be tried, although in the presence of fever it is often unavailing.[1]

Pericarditis may complicate both chorea and arthritis and is probably the most serious cardiac lesion, tending to leave the pericardium adherent and thus gravely disabling the heart. In the extremely acute cases, which appear to be going from bad to worse, the author has found 20 c.c. of concentrated streptococcus antitoxin (scarlatina) serum (Burroughs, Wellcome & Co.) occasionally most helpful, and this is always worth administering, especially if it can be given early and in large repeated doses.

Adherent pericardium.—In an effort to compensate for this disability the heart hypertrophies to a great extent, and in fact at post-mortem in cases of rheumatic carditis the *cor bovinum* is frequently seen. Clinically, adherent pericardium may be suspected by the size of the heart and the fact that it has been preceded by pericarditis, but in addition the inspection of the intercostal spaces in the left axilla and at the angle of the scapula will often show a dragging in and out of the chest-wall with each cardiac impulse.

The outlook in cases of adherent pericardium is extremely grave, most of the patients ultimately becoming hopelessly crippled or dying at an early age.

Care of a rheumatic child with involvement of the heart.—Just as in the typical case of chorea described at p. 447, the child should be kept lying

[1] George Sutherland, "The Therapeutic Action of Digitalis on the Rapid, Regular Rheumatic Heart," *Quart. Journ. Med.*, Vol. 12, No. 47.

perfectly flat, and if necessary restrained in this position by means of shoulder-straps. The pulse should be charted four-hourly or at least twice daily (Figs. 76 and 77). Sodium salicylate should be given along with bicarbonate of soda in doses of from 20 to 40 grains daily.

The diet should be, as in chorea, light and nourishing, yet one not likely to make the child bilious (*see* p. 134). It should be pushed just short of the limit of the child's digestion.

If the pulse-rate drops rapidly with rest, no murmurs are present, and the dilatation of the heart rights itself. A little more strain may now be thrown on the heart. A second pillow, then a third, then a back-rest may be allowed; later the child should sit up and feed himself, and finally may get up and slowly resume his ordinary life. The pulse-rate, however, is the all-important factor in controlling this resumption. At any sign of a rise of pulse-rate or of the appearance of a murmur or fresh cardiac damage the child should be made to revert to the flat position. This state of absolute rest may have to be maintained for many weeks if the cardiac damage is considerable. The parents should be instructed to take the child's weight at not longer than monthly intervals, and at any sign of loss, fresh limb-pains, increased shortness of breath or pallor, a recurrence may be expected and medical aid should be sought. To get the child well nourished and healthy in appearance and keep him in such a condition is the only safeguard against a future attack.

After-care.—Open-air schools and prolonged convalescence under the care of persons who understand the problem should be advocated. Septic teeth and tonsils should be dealt with in every case as soon as the active signs of rheumatism have sufficiently subsided. Just as in adults, the gauge as to what a child should be allowed to do is an individual one. By slow degrees more and more strain may be thrown both on the child's heart and on his whole mode of living. If he continues to gain weight, and his cardiac compensation continues to be satisfactory, there can be no harm

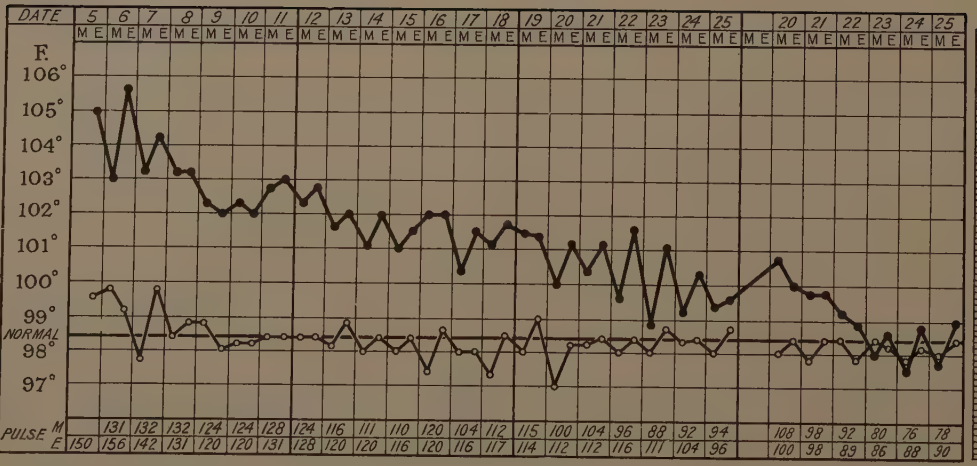

Fig. 76.—Temperature and pulse chart in a case of rheumatic carditis. The upper line shows the pulse-rate and the lower line the temperature.

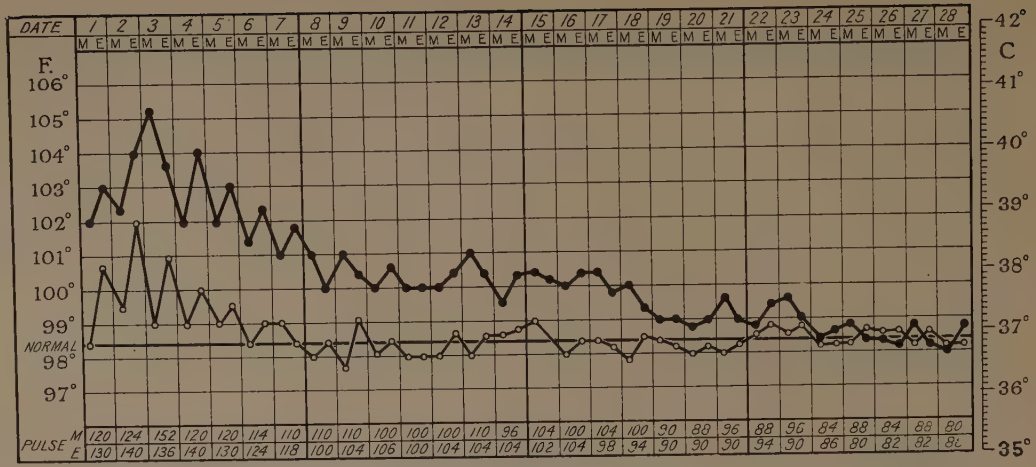

Fig. 77.—Temperature and pulse chart in a case of rheumatic carditis. The upper line shows the pulse-rate and the lower line the temperature.

in letting him resume a normal life, but at any sign of a breakdown in either, retrenchment must be imposed and a resumption should be made on much slower and more cautious lines.

Prognosis.—With efficient care the mortality is extremely low in all types of rheumatism, probably not exceeding 5 to 10 per cent. The proportion of complete recoveries is about 50 per cent., and the remainder are either moderately or grossly disabled. If treatment is not followed up the mortality in any given group of cases rises steeply, almost all the severely damaged or untreated cases succumbing at an early age.

How to Prevent Rheumatism

The most effective method of prevention is to keep the child thoroughly fit. If he has bad teeth or septic tonsils they should be attended to early, so that he is never allowed to get run down. He should be weighed at intervals, and at any failure to gain weight over a considerable period he should be examined carefully for organic disease, and his diet and home régime carefully investigated to ascertain where the error is being made. Damp districts and damp houses should be avoided, and the child must not be allowed to put on damp underclothing, or wear damp underclothing after he has exerted himself and perspired. After games he should be given an opportunity to change his clothes completely, especially the underclothes. His diet should be a general, all-round, well-balanced one containing meat, not a faddy, meatless one.[1] The wealthy eat much meat and have no rheumatism; the poor eat very little and rheumatism is peculiarly their disease.

At any sign of "growing-pains," pallor or anæmia, lassitude, sore throat, undue nervousness or emotional instability, the child should be carefully examined and steps taken to deal with such symptoms, as they may be the earliest manifestations of rheumatism.

[1] A. P. Thomson, "The Effect of Variation of the Diet in Rheumatic Children," *Arch. Dis. Child.*, Vol. 4, No. 21, p. 152.

Rheumatoid Arthritis (Still's Disease) [1, 2]

This condition in childhood was very fully described by Dr. G. F. Still in 1897 [3] and very little has been added to his original description.

Etiology.—The affection is slightly commoner in girls than in boys, and the majority of cases commence in the second, third or fourth year of life. The cause is not definitely known, but it is usually thought to be due to an infection gaining entrance either through the tonsils or from some septic focus in the sinuses or elsewhere. Bacteriological examination of the periarticular tissues and of any effusion into the joints has repeatedly shown these to be sterile.

Pathology.—The chief changes are periarticular, and the joints most commonly involved are the knees, ankles, wrists, elbows and the interphalangeal joints. Every joint in the body, however, may be involved, and commonly the vertebræ are grossly affected. With disuse and the accompanying general ill-health of the child, there is marked osteoporosis of all the bones, a condition which is revealed by X-ray examination. There is general enlargement of the glands, the spleen being involved occasionally, but the epitrochlear glands are invariably swollen, inguinal and axillary glands also being prominent. In a case of long standing the synovial membrane is thick and gelatinous and the cartilage becomes more and more eroded and involved until finally it shows gross destruction, with partial or complete ankylosis of the joint.

Clinical picture.—A child aged two or three has an acute feverish illness, the joints rapidly becoming swollen and very tender. Fever is not always a prominent feature, however, and may have escaped notice. Movement is limited at the elbows, wrists, fingers, knees and ankles. The head is moved with difficulty, and chewing may be impeded because of pain

[1] F. J. Poynton, in Garrod, Batten, Thursfield and Paterson's "Diseases of Children" (1929), p. 901.
[2] H. Rolleston and H. A. T. Fairbank, *ibid.*, p. 861.
[3] G. F. Still, *Med. Chir. Trans.*, London, 1897, Vol. 80, p. 47.

or stiffness in the temporo-maxillary joints. The appearance of the various joints is typical, there being a fusiform swelling about the joint with wasting of the muscles above and below (Fig. 78). Although pain is often a feature at the very onset, the joints rapidly become much less painful, finally becoming practically painless unless overmanipulated. At intervals there is an acute fever for a day or two, with malaise and a return of pain to the joints; at the same time the glands become slightly more enlarged and tender, and then again subside.

Fig. 78. Rheumatoid arthritis. Note obvious involvement of the knees, ankles and wrists.

Differential diagnosis.—Because of the stiffness of the neck, spinal caries must be ruled out by X-ray examination. The chronic joint involvement excludes acute rheumatism, and this inference is supported by the fact that the heart is not involved. Syphilitic arthritis as a rule involves the knees only, and the Wassermann reaction serves to exclude syphilis.

Prognosis.—On the whole the prognosis must be considered extremely bad, many cases succumbing to intercurrent infections. The author has seen very

few cases completely recover under any form of treatment at present known.

Treatment.—The removal of septic foci, such as infected teeth, tonsils or sinuses, should first be undertaken. The author has seen a number of cases running a continuous fever in which the temperature subsided with an accompanying improvement in the joint condition after tonsillectomy. Local treatment to the joints, such as massage, passive movements, artificial sunlight and radiant heat, is very useful and should be undertaken. Cases have been reported in which, after acute infectious diseases such as scarlet fever and septicæmias, especially streptococcal septicæmias, a complete cure occurred. This suggests some form of protein shock or anaphylaxis. In the author's experience more hope lies in treatment along such lines than in any other direction. Protein shock[1], induced with T.A.B. (typhoid, paratyphoid A and B) vaccine, sterile milk, or peptone injections given intravenously, have all been employed successfully. In one such case, where following on tonsillectomy the child developed a septicæmia, on this subsiding all signs of the rheumatoid arthritis rapidly disappeared. This child was seen one year later and appeared perfectly healthy.

Where gross deformity is present, orthopædic treatment must be undertaken. Special boots and irons prevent further contractions and deformity.

No special diet is required, but tonics such as cod-liver oil and iron are indicated.

[1] J. H. Thursfield, "Notes on a Case of Chronic Polyarthritis (Still's Disease) Treated by Intravenous Protein Therapy," *Arch. Dis. Child.*, Vol. 2, p. 175.

CHAPTER XIX

DIABETES

Diabetes Mellitus

Etiology and pathology.—There are two possible explanations of the occurrence of diabetes in childhood : first, that there is a congenital deficiency of the islands of Langerhans, and secondly, that there is a damage to the existing islands of Langerhans by poisons or infection. At post-mortem, even with the most careful examination by our present methods, fibrosis or deficiency of the islands can only be shown in a minority of the cases. In view of the fact that the administration of insulin, a product prepared from the islands of Langerhans, appears to be specific in the removal of the excess of sugar from the blood, causing it to be metabolized, we must assume in our present state of knowledge that diabetes is due to a dysfunction of this part of the pancreas.

Diabetes is slightly more common in males than in females. In a small proportion of cases it is hereditary.

Clinical picture and diagnosis.—The disease may commence at any age, but is seldom diagnosed in children below the age of two. The earliest age at which it is usually recognized is 5 or 6 years. The child is noted to be losing weight rapidly, to have an intolerable thirst and to be passing water too frequently and in excessive quantities. Enuresis may often attract attention to the child's condition. Lately the author has seen two cases commencing after severe sore throat, and improving after tonsillectomy to the extent of requiring no insulin. An examination of the urine

shows the presence of a reducing substance, which is glucose, and accompanying this there may be diacetic acid and acetone. On examination of an untreated diabetic, the tongue may be red and clean, the skin dry and the palms of the hands and soles of the feet slightly yellow. A sweetish acetone smell is often present in the breath. If the condition is severe and has been untreated, peripheral neuritis may be present to such

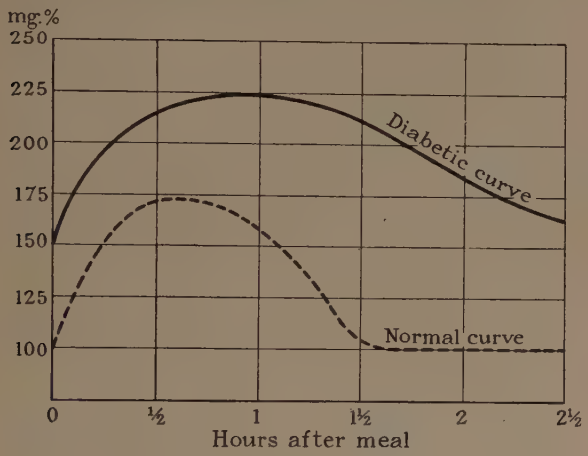

Fig. 79.—Blood-sugar curve of a normal child and of a diabetic child.

an extent that the knee-jerks are lost. The child is rapidly reduced to an extreme degree of emaciation.

Blood-sugar curve.—An examination of the blood-sugar of a normal child shows that the resting blood-sugar stands at about 100 mgm. per cent. After a meal or the ingestion of glucose it rises rapidly to 175 mgm. per cent. within half an hour, and within one and a half hours has fallen to normal or nearly so, remaining down. The blood-sugar curve of a diabetic, on the other hand, not only starts higher, say 150 mgm. per cent., but after the ingestion of sugar or a meal it rises steadily to 200 or 225 mgm. per cent. and may even

go much higher or remain there for an hour or more, settling much more slowly than the normal (Fig. 79).

From this it may be seen that the normal sugar-content of the blood in diabetic is much higher than in non-diabetic children and, being above the sugar-threshold of the kidney, leads to a constant leaking through of sugar into the urine.

Renal glycosuria.—Occasionally children are found who have sugar in the urine yet whose sugar-tolerance curve and blood-sugar turn out to be quite normal. On the other hand they have a low threshold for sugar, allowing it to pass out into the urine at a lower level than normal.

Diabetic coma.—A child with diabetes may at any time develop coma, and this condition may set in rapidly. If the proportion of fats is relatively too high, so that the metabolism is incomplete, allowing acetone and diacetic acid to collect in the blood, the symptoms of coma appear. Yawning, drowsiness, irritability and vomiting may herald the onset. Later, air-hunger is marked and complete unconsciousness occurs. A too-liberal supply of fat, or a sudden infection may induce coma, and the closest observation of the diabetic child is therefore necessary.

Hypoglycæmia.—If too large a dose of insulin is given the child becomes pale and lifeless, occasionally he is irritable and extremely emotional. He is seized with shakiness of the hands and giddiness, and if the condition is not checked severe convulsions with unconsciousness ensue. For treatment *see* pp. 470, 471.

Prognosis.—Previous to the introduction of insulin the life of the diabetic was short. Now it appears that, with care, life can be prolonged indefinitely. The child puts on weight (Fig. 80), develops, and appears normal, mentally and in every other respect. The dose of insulin may be slightly reduced, and it is held by some observers that the pancreas shows a tendency to regenerate. In other cases, however, there is a steady tendency downhill, a larger and larger dose of insulin being required to maintain the body-weight.

Treatment. General management of the child.—The child should have his breakfast in bed, the meal being immediately preceded by the dose of insulin. With a child aged five or more it is best to start kindergarten or systematic play, so that he can be employed sitting down throughout the morning. A gentle walk

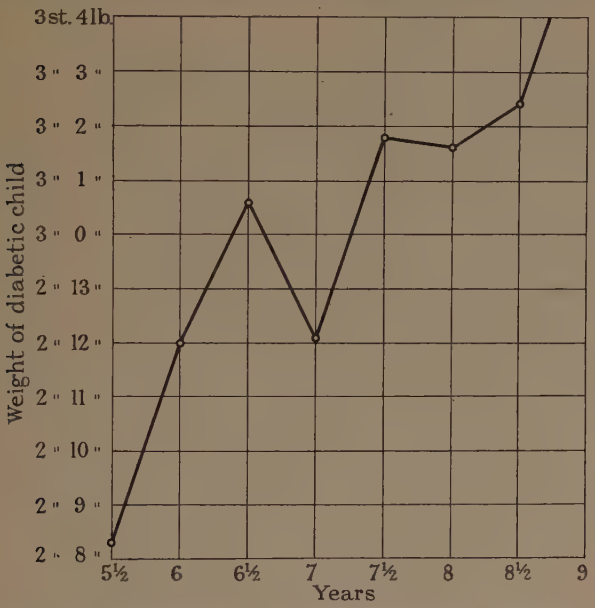

Fig. 80.—Weight chart of a child under treatment for diabetes.

or outing in a push-chair completes the morning régime. The insulin is given immediately before dinner, which is a substantial meal, and an hour's rest follows immediately after. The child is then sent out to walk, after which a small tea is given at about 4 o'clock, and this is followed by a romp. Supper is given between 6 and 7 o'clock and the child is put to bed within an hour.

Diet.—A sighting shot must be made in regard to diet, and for this purpose the above table will be found useful. Then sufficient insulin should be given to control glycosuria. It may be given once, twice or even three times in the day. It will be found best as a rule to start with half the quantity of fat mentioned in the table.

TABLE XV [1]

FOOD REQUIREMENTS OF A DIABETIC CHILD

Age in years.	*Ideal wt.* Kg.	lb.	*Calories per kg. body-wt.*	*Calories per diem.*	*Gm. protein per kg. body-wt.*	*Daily intake in grammes.* *Carbohydrate.*	*Protein.*	*Fat.*
1	9	20	100	900	4	50	35	60
2	12	26	90	1050	3.5	60	40	70
3	14	31	80	1150	3	60	45	80
4	16	35	80	1300	3	60	50	90
5	18	40	80	1400	3	60	55	100
6	20	44	75	1600	3	60	60	120
8	24	53	75	1800	2.5	70	60	140
10	28.5	63	70	2000	2	75	60	160
12	34	75	60	2100	2	80	65	160
14	43	95	50	2100	1.5	80	65	160

In treating a case of diabetes a good book of food-tables such, for example, as that of Harrison and Lawrence,[2] is almost a necessity. A suitable menu for a diabetic child for one day is appended (*see* Table XVII, p. 470). It will be found most helpful to attempt to keep the protein low and to allow as much carbohydrate as can be controlled by a reasonable dose of insulin. The fat should be pushed to a point just short of the appearance of diacetic acid in the urine. The author is of the opinion that less protein is advisable than that given in Harrison's schedule (*see* Table XV), and that it is unlikely that the total of the fat suggested

[1] G. A. Harrison, in Garrod, Batten, Thursfield and Paterson's "Diseases of Children" (1929), p. 540.

[2] G. A. Harrison and R. D. Lawrence, "Food Tables" (Skinner & Co.).

can be reached. A teaspoonful of Scott's Emulsion should be given three times daily.

Testing the urine.—Every specimen of urine passed —there may be six or even eight specimens per diem— should be tested for diacetic acid and sugar, and the insulin raised or lowered until the output of these two has been reduced to a minimum. In this way it will be found after which meals the most sugar is present, and then an appropriate dose of insulin can be given to control the sugar at these times on the following day. The best *test for diacetic acid* is the addition of iron perchloride to the urine, which, if diacetic acid is present, assumes a dark port-wine colour. The fallacy in this, however, is that the test is positive when salicylates are being given, and in this case the urine must be tested for acetone by the *Rothera test*, as follows: A few drops of freshly-prepared sodium nitro-prusside solution are added to the urine, followed by ammonia until it is alkaline, and then the liquid is saturated with crystals of ammonium sulphate. If acetone is present a deep-violet colour develops in from one to fifteen minutes.[1] *Sugar is tested for* by filling up with Fehling's solution 1 and 2 in the usual way. It is only by getting a specimen before each meal, i.e. before giving insulin, and testing each carefully, that hypoglycæmic attacks can be avoided and the insulin successfully regulated. As a rule diabetics wake once in the night, and this night specimen of urine should be kept and tested. The record of a typical day in a diabetic patient's life is given on p. 468. Whenever diacetic acid appears, the indication is to reduce the fats drastically. If much diacetic acid is present, it may be necessary to give additional sugar and insulin at the same time. The examination of the blood-sugar, although of extreme importance in diagnosis, is not, in the author's opinion, of much practical help in the treatment of the case. The careful examination of the urine at frequent intervals is the sheet-anchor.

[1] Hutchison and Hunter, "Clinical Methods," 9th Edit. (1929), p. 380.

TABLE XVI

RECORD OF A DIABETIC CHILD FOR ONE DAY

Date.	*Time.*	*Sugar.*	*Diacetic acid.*	*Grammes of food.* Carbo-hydrate.	Protein.	Fat.	*Insulin.*
16.11.27	12.15 a.m.	Nil	Nil				11 units
	7.45 „	++	Nil				8 a.m.
	9.0 „	+++	Slight				11 units
	12.30 p.m.	Nil	Nil				1 p.m.
	4.0 „	Nil	Nil				9 units
	6.0 „	Nil	Nil				7 p.m.
	7.30 „	Nil	Nil				*Total* 31 units
	8.30 a.m.	Breakfast		17	14½	29¾	
	1.0 p.m.	Dinner .		31½	16¾	35	
	4.0 „	Tea . .		8	13½	24	
	7.0 „	Supper .		19	4	10½	
				75½	48¾	99¼	

Technique of administration of insulin.—Actually, in a moderately severe or severe case of diabetes it is much easier to keep the patient free from sugar on three doses of insulin daily than on two, and the prick of the needle is soon borne extremely well by the child if the right attitude is adopted by those attending him. The inconvenience involved in giving insulin should be considered as nothing compared with the benefit which accrues from it. A different part of the body should be chosen at each administration, i.e. in the morning the arm may be used, at midday the thigh, and in the evening the buttock, all on the same side of the body. The following day this procedure may be repeated, but on the opposite side of the body. The syringe and needle should be boiled at each administration, as fine a needle as possible being obtained. The skin should be iodined and also the top of the insulin bottle. If indurated areas appear after injections, these places should be avoided until they have entirely recovered.

Errors in the management of the diabetic child.—There are certain gross errors which are liable to be made in calculating any diet.

1. The composition of the various foodstuffs may obviously *vary greatly*. For example, the difference in food-content between a bitter and a sweet orange, a ripe and an unripe apple, old and new potatoes, fat and lean meat, etc., is a great source of error.

2. The *presence or absence of infections* must be considered. A slight tonsillitis or a cold burns up the available blood-sugar very quickly, and in diabetes there seems no power to store sugar, thus there is no reserve. The fat is therefore unmetabolized, resulting in acute ketosis, or, if fat is present in small quantities only, the symptoms may be those of acute hypoglycæmia. On this account it is most important that all sources of sepsis be investigated in the diabetic child. The removal of septic tonsils or teeth should be carried out, and it will be found that this can easily be done if care is taken. For 24 hours before the anæsthetic is given the fat should be restricted, a small amount of sugar being allowed to appear in the urine in order to ensure against a ketosis following anæsthesia.

3. The *action of aperients* must be taken into account. If the bowels move daily naturally, a certain standard of food-absorption efficiency is established. If the child becomes slightly constipated, the food is more perfectly absorbed, but on administering a purge, even of a mild character, a great wastage must take place, and attacks of hypoglycæmia threaten to occur as a result. Probably the best laxative is simple liquid paraffin, given night and morning, but if a saline is required, Seidlitz powders, which contain no sugar, will be found most suitable.

4. The *amount of exercise* taken in the day is important. With much exercise more of the blood-sugar is used up. Less insulin need then be given, and consequently less sugar is available for fat metabolism. Suppose a child getting, say, 11 units of insulin before dinner daily, and having a very quiet morning, is allowed to romp and play strenuously with other children for an hour preceding this meal on a particular day. This unusual occurrence will produce

a marked hypoglycæmia, and if this romp could have been anticipated less insulin need have been given. It will be apparent, therefore, that absolute regularity in the amount of exercise and excitement is necessary if the dose of insulin is to be the right one and attacks of ketosis or hypoglycæmia are to be avoided.

TABLE XVII [1]

A Day's Menu for a Diabetic Child

		Weight in oz.	*Calorie value in grammes.* Carbo-hydrate.	Protein.	Fat.
Breakfast.	Fried bacon	1	—	5	15
	Fried egg		—	6	5½
	Bread fried in bacon fat	⅛	5	1	5
	Pears stewed without sugar	1½	2½	—	—
	Force	¼	5	½	—
	Milk	3	4½	3	3
	Coffee to drink				
			17	15½	28½
Dinner.	Raw beef (Beef stew)	2	—	10	4
	Butter (Beef stew)	⅝	—	—	10
	Carrots (cooked) (Beef stew)	1	2½	½	—
	Turnips (cooked) (Beef stew)	2¼			
	Milk (Ground rice pudding)	6	9	6	6
	Ground rice (Ground rice pudding)	⅝	10	—	—
	Butter (Ground rice pudding)	⅝	—	—	15
	Apples stewed without sugar	5	10	—	—
	Cream	1	—	—	5
			31½	16½	40
Tea	Pressed beef	1½	—	10	12
	Bread	½	5	1	—
	Butter	⅝	—	—	10
	Milk	2	3	2	2
			8	13	24
Supper.	Bread	½	5	1	—
	Butter	⅝	—	—	10
	Energen bread	½	4	2	½
	Orange	3½	10	1	—
			19	4	10½
	Total for the day		75½	49½	103

[1] From Paterson and Forest Smith's "Modern Methods of Feeding in Infancy and Childhood."

Treatment of acute hypoglycæmia.—Adrenalin, ½ c.c., should be given hypodermically if the child is in a state of convulsion and cannot swallow. Glucose, preferably in solution, should be given rectally immediately following the adrenalin. If the child is in the earlier stages of an attack lump sugar given by the mouth will rapidly stop the trouble. From 4 to 8 lumps should be given and the effect noted. It is well to ascertain the cause of the hypoglycæmic attack by a scrutiny of the possible sources enumerated above. Thus further attacks can be foreseen and prevented.

CHAPTER XX

INTESTINAL PARASITES (WORMS)

There are three worms commonly found parasitic in infancy and childhood in the British Isles. These are :

(1) Tænia mediocanellata (tapeworm),
(2) Ascaris lumbricoides (roundworm), and
(3) Oxyuris vermicularis (threadworm).

Tænia Mediocanellata (Tapeworm)

This is a relatively common intestinal parasite, but the source of infection is very often difficult to ascertain. Occasionally a dog or cat has been found to be the infecting agent, and in some cases the patients or their parents will be found to have recently returned from the East. The age-incidence shows that this form of tapeworm is commonest between the ages of 2 and 8 years. The worm varies in length from 5 to 9 yards, and is segmented, the segments being yellowish white, flat and, in the centre of the worm, broader than they are long, whereas towards the head and tail they are longer than they are broad. As the head is approached, the segments rapidly become elongated and very slim, and in the last few inches are rarely more than $\frac{1}{8}$ inch across, although they are about $\frac{1}{4}$ inch long. The head itself is no larger than the head of a pin, and the neck leading from it is about the thickness of a pin. Thus, the finding of the head and neck, though a matter of great importance, is also a matter of extreme difficulty. The worm fastens itself with its head beneath the folds of the bowel high up. Its site probably varies, however, though in the most difficult cases it is usually no great

distance from the duodenum. The lower down the bowel it is the more easily can it be dealt with.

Symptoms and diagnosis.—It may be noticed that the child is wasting, or is suffering from bouts of colitis, passing large, mucus-containing stools. Some mothers claim that the colour of their children is yellowish or earthy during the presence of the worm. Certainly they are not as good a colour at this time. Then segments may be noted appearing in the stools, either singly or two or three segments fastened together, each probably the size of an average finger-nail and somewhat similar in shape. This means that the presence of a tape-worm is unmistakable and the diagnosis is a simple one. Segments are passed in almost every motion and occasionally are found in the morning in the bed.

Treatment.—Once the diagnosis has been made, treatment should be commenced. It is important to follow the details rigidly, the treatment given being suitable for a child aged 5 to 7 years.

The first day.—The diet is light and one which leaves as little residue as possible. For breakfast, groats, porridge or other cereal, accompanied by some sweet preparation, such as syrup or honey, is suitable, and weak tea or skimmed milk should follow. At midday the child should have soup thickened with potato, but green vegetables should be avoided on account of the residue. A little ground-rice pudding should follow the soup. Tea should consist of more cereal or thin bread and butter, with honey or syrup, and weak tea or skimmed milk to drink. At night a brisk purge of grey powder, cascara, syrup of senna or syrup of figs should be administered.

The second day.—The diet is the same and at night a similar purge is given.

The third day.—The treatment is now to be carried out. At, say, 7 a.m. the child may have a little weak tea with a lump of sugar in it, but nothing more. At 9 a.m. a capsule containing 15 minims of extract of filix mas should be given, and this dose repeated at half-hourly intervals until four doses have been given.

(Should the first treatment have failed, 6 doses may safely be given during a subsequent treatment, provided the after-treatment is rigidly adhered to.) This means that the fourth capsule has been given at 10.30 a.m. At 11 a.m. a tablespoonful of castor oil is administered and the worm may be expected within an hour. Black crêpe is placed over the chamber and the motion passed directly into it. This is then washed through and the white segments of worm stand out plainly on the black crêpe and are easily distinguished. As a rule the larger portion of the worm will be found to be passed unbroken, and by following it up it is quickly disposed of. A search should then be made among the short slim portions for the head.

Should the treatment fail it must not be repeated for from six to eight weeks, and then, as previously stated, a slightly larger dose, that is, up to a total of 90 minims, is permissible, provided the castor oil is given and retained, and acts promptly. A total of 60 minims is safe for the age of, say, 2½ to 3 years. Some cases are very resistant, and in one recent case the worm was finally removed only on the fifteenth treatment, and then only by a combination of filix mas and chenopodium. Before giving the capsules they should be held in warm water until they are very soft and thin. In this way their contents are available high up in the small intestine.

Oil of chenopodium treatment.—Here there need be no preliminary fasting. Give the patient 2 teaspoonfuls of aromatic cascara the night before beginning the treatment. If no bowel movement results give a soap-suds enema in the morning. Omit breakfast. Give ½ to 1 drop of the oil of chenopodium per year of the patient's age on a lump of sugar or bread at 8 a.m., i.e. 4 to 8 drops of the oil of chenopodium if the child is 8 years of age. Repeat the same dose at 10 a.m. Repeat the same dose at 12 noon. Do not exceed this dose. Omit lunch. Give 1 to 2 tablespoonfuls of castor oil at 12.30 p.m. (i.e. half-an-hour after the last dose of chenopodium). Give the usual supper. This course may be

repeated after an interval of one week if there is any doubt of the efficacy of the first course.

Tænia cucumerina.—This small tapeworm, from 6 inches to 12 inches long, is occasionally present in infants. One infant, aged six months, was treated with $\frac{1}{10}$-grain doses of calomel successfully.

Ascaris Lumbricoides (Roundworm)

The host of this worm is as a rule the dog or cat, the ova being carried to the mouth on the child's fingers and swallowed after caressing the animal. A fully developed worm is 6 to 8 inches in length, and except for the fact that they are much larger, they resemble the ordinary earthworm seen on a golf course.

Children infected with ascaris appear in moderately good health. Restlessness is complained of, loss of appetite and undigested and mucus-containing stools, but the patients are on the whole comparatively well nourished notwithstanding. Worms are not present in large numbers, an occasional one or two being passed at infrequent intervals. At times the worms are produced from the child's mouth. This migration is dangerous because of the possibility of respiratory inhalation. The worms may be found also in the child's bed, or may be passed by themselves without any motion.

Treatment.—The diet should be similar to that described for tapeworm, namely low in protein and fat, and high in carbohydrate, so as to leave very little residue.

A mixture, containing santonin $2\frac{1}{2}$ grains, compound scammony powder 2 grains, calomel $\frac{1}{2}$ grain, should then be given at bed-time for three nights, and first thing in the morning a tablespoonful of milk of magnesia or a teaspoonful of Eno's Fruit Salts should be administered.

Care should be taken that no oily substance such as castor oil or olive oil is given during this period, otherwise santonin-poisoning, with coloured vision and vomiting, is likely to occur.

Oxyuris Vermicularis (Threadworm)

Cases of infection with threadworms are common, especially among the hospital classes, and girls are more

commonly infected than boys. Threadworms are white in colour, from $\frac{1}{4}$ inch to $\frac{1}{3}$ inch in length, and of about the thickness of a piece of fine cotton thread. They are present in large numbers in the motions, the ova being passed at the same time as the threadworms. The child is infected by swallowing the ova derived from infected persons or domestic animals, and the infection is often kept going by the child re-infecting itself; the ova, getting beneath the finger-nails and being conveyed to the mouth, pass through the stomach into the small intestine, where they hatch. It is the opinion of the author that most people are constantly ingesting the ova of worms. Those who are below par and run down have a low hydrochloric acid content and therefore fail to digest and kill off the swallowed ova. The symptoms of threadworm infection therefore suggest rather that the child is run down than that the worms are really harmful. It is very common in families to find one child with worms and the remainder free, although all live in close proximity and have the same opportunity of becoming infected. The ova do not hatch in the colon where they are produced. The outside cellulose covering must be digested off before they can perform their life-cycle, and it is because conditions are ideal in the debilitated child that this development is able to take place.

One of the commonest **symptoms** is irritation about the anus and vulva, and cases of vulvo-vaginitis can frequently be traced to this continuous irritation set up by worms. Picking at the nose and crying at night are not necessarily indicative of infection with worms, as was formerly thought. Worms may be seen protruding from the anus, either in the daytime or at night, as fine threads, or they may be found in the bed. When a motion is passed the worms are motile and, as a rule, easily seen, but sometimes particles of food débris, especially from bananas and cabbage, are mistaken for threadworms. The infected child very often shows pallor, loss of weight and poor appetite. There are often recurrent attacks of mucous colitis, and occa-

sionally at operation the appendix is found plugged with the worms.

Treatment.—1. The nails should be clipped short, so that the ova cannot lodge there. The hands should be scrubbed several times daily and care must be taken to prevent them from being contaminated with fæces.

2. A weak white precipitate ointment should be applied to the anus morning and evening in order to kill off extruding worms or their ova, and allay irritation.

3. Gloves should be put on the hands and suitable pyjamas or other night clothing worn to prevent the child from scratching itself during the night and thus conveying the ova to the mouth.

4. A daily enema of infusion of quassia and water, equal parts, about 4 to 6 oz., should be given.

5. Probably the most suitable preparation by the mouth is Butolan (Bayer Products, Ltd.), half a tablet three times a day for one week. During this week a brisk purge should be given on alternate nights, such as 3 or 4 grains of grey powder at bed-time, with a dessert-spoonful of milk of magnesia the following morning.

6. As the debilitated child furnishes good soil for threadworms, treatment along general lines, such as the removal of septic teeth and tonsils, plenty of fresh air and a well-balanced diet, with cod-liver oil and malt, is strongly indicated. Frequently in threadworm infection a fortnight at the seaside is the most efficient anthelmintic.

7. As a tonic the most suitable mixture is the following:

Pepsin, 4 grains.
Acid. hydrochlor. dil., 8 minims.
Syr. senn., 10 minims.
Aq. chlorof. ad 1 drachm.
Two drachms in a cup of water with meals.

By raising the gastric acidity the ova of the threadworms are destroyed.

CHAPTER XXI

SYPHILIS IN CHILDHOOD

In the vast majority of cases of syphilis in childhood infection takes place before birth. These are termed cases of congenital syphilis. In rare cases, however, syphilis is acquired after birth (extra-genital infections).

Congenital Syphilis

Etiology.—Syphilis is the result of an infection by the *Spirochæte pallida*. In most cases the father has contracted syphilis and has passed on the infection to the mother, who gives birth to a syphilitic infant. It is an interesting fact that very often the mother shows no clinical signs of the disease, although she may have a positive Wassermann reaction. It is argued by some that this is because she has harboured a syphilitic placenta and has thus developed immune bodies. The mother, then, may become infected by a primary vaginal infection or through an infected placenta. It is assumed that the ovum is fertilized by an infected spermatozoon. In new-born infants the Wassermann reaction may be applied to the mother in addition to the child, but as a rule if it is found positive in the one it is positive in the other also. Although the result of the Wassermann reaction in the father may be found negative, this does not disprove that he may have been the primary infecting agent, as he may have had treatment subsequently to infecting his wife.

Syphilitic infants are not highly infectious to other children. There is no doubt that infection can be carried, but instances of the disease being acquired in this way would appear to be extremely rare. The suckling of a

syphilitic infant by a wet nurse is likely to produce breast infection, but the mere handling of the child is not likely to lead to infection.

Clinical picture.—A syphilitic infant may be *born dead*, either at full term or prematurely. It may be a *macerated fœtus*, the placenta being infected, friable and containing infarcts. A history marked by such events as the following is quite common: first, an abortion at 2 or 3 months; then possibly another at 5 months; then a premature birth or stillbirth at 7 months; and finally a full-term child apparently quite healthy. At birth as a rule the child shows no signs of syphilis. In a very small percentage only the *rash* is present at birth or develops within the first week; in the great majority it develops between the fourth and eighth weeks. The rash may be a *macular* one, which appears on the buttocks, the palms of the hands, the soles of the feet and the legs; it is pale-red or copper-coloured, and has a shiny, dry, silky appearance. It may be present over the whole body, but this is uncommon. The syphilitic *pemphigus* which occurs on the soles of the feet, palms of the hands, the knees and sometimes the face, does not usually appear for two or three weeks, but occasionally the infant is born with it. There may be a *syphilitic wig* present, that is, in congenital syphilis the hair may be thick, black and straight, but this is of no great importance, as many normal infants have a profuse growth of hair. *Snuffles* is one of the earliest symptoms to appear. It may be present at birth or may appear within a few days, or may develop after some weeks. Snuffles is due to a syphilis of the septum and turbinate bones, in which other organisms take part, causing a muco-purulent discharge and destruction of the nasal bones. The bridge of the nose then falls in and the flat, saddle-shaped nose results. *Rhagades*, or scarring about the mouth in its most typical form, is best seen in older children. In the younger child it assumes the picture of sores about the mouth or merely roughness. *Condylomata* form at the angles of the mouth and anus (Fig. 81), and on healing leave

permanent scars. In older children a *syphilitic perforation of the soft palate* (Fig. 82) may occasionally be seen. In little infants with syphilis the cry is often hoarse, due to a syphilitic laryngitis. In about half the cases there is an *enlargement of the spleen* within a few weeks of birth. A new-born infant with an enlarged spleen, whether it has any of the other manifestations of syphilis or not, is a syphilitic suspect. Combined with other manifestations of syphilis, this symptom renders the diagnosis almost certain.

Fig. 81. - Congenital syphilis, showing condylomata about the anus.
(By courtesy of Dr. Nabarro.)

Syphilis of the bones.—One of the earliest manifestations of syphilis is that of Parrot's pseudo-paralysis. Here the child develops acute syphilitic epiphysitis, usually at the upper end of the humerus, but it may be elsewhere. It is noted that the arm is not used, and the question is raised whether a fracture may have occurred at birth unnoticed. As a rule there are other manifestations of syphilis, however, which help in the diagnosis. The X-ray appearance is typical (Plate 16), showing an enlargement of the upper end of the humerus, with a marked osteoporosis. The commonest lesion in

Plate 16.—Radiogram showing syphilitic epiphysitis. Note also the well-marked periostitis.

congenital syphilis is osteitis, best seen in the tibiæ, which show bowing and curving forward, and in the thickening of the skull-bones, especially the bossing over the frontal and occipital regions. A syphilitic dactylitis shows a typical periostitis in the skiagram.

Syphilitic meningitis.—Cases of syphilitic meningitis as a rule manifest themselves by hydrocephalus,

Fig. 82.—Syphilitic perforation of the palate.

which is usually external. The head becomes larger and larger, the spleen may be palpable, and the child may show other signs of syphilis. The Wassermann reaction applied to the child will show the blood or cerebro-spinal fluid to be positive. It is always well to test the mother at the same time. The post-mortem appearance of syphilitic meningitis shows much thickening of the meninges, with a tendency to the formation of

adhesions and of cystic spaces into which hæmorrhages may take place. As a rule the diagnosis of such cases is merely hydrocephalus. The choroid plexus, functioning normally, has produced the cerebro-spinal fluid, but the inflammation of the meninges has prevented the fluid from draining away, resulting in hydrocephalus. The fluid may be blood-stained, but as a rule it is moderately clear. The albumin is raised, sugar is present in normal amount and lymphocytes are increased.

Lesions of the eyes.—The commonest lesion is choroiditis, when black or dark patches may be seen scattered over the choroid. This lesion is present at any age. Interstitial keratitis is a lesion coming on as a rule after the commencement of the second dentition, between the ages of six and twelve. With its photophobia, opacities of the cornea and accompanied by peg-shaped teeth or some other manifestation of syphilis, the diagnosis is comparatively easy.

Mental deficiency.—A certain small proportion of congenital mental defects are due to syphilitic infection. The vast majority of these, however, are hydrocephalic.

Syphilitic testis is rare. A Wassermann test should always be employed in every gross enlargement of the testis in childhood.

Along with other manifestations of syphilis a *hard, enlarged liver* may be felt. There may be two processes at work: (*a*) miliary gummata, with fine, intercellular cirrhosis, (*b*) localized patches of gummatous material, very often just beneath the capsule. It is said that the left lobe of the liver is more often involved in syphilis of childhood than the right. Extreme jaundice due to syphilis is a great rarity in childhood.

In rare cases one meets with *syphilitic carditis.*

White pneumonia in the new-born infant is also extremely rare. In such cases there is an interstitial fibrosis of syphilitic origin, involving the whole of one lung.

Syphilitic nephritis.—This is the commonest cause of œdema in infancy. It comes on as a rule between the

fourth and the eighth month and often proves fatal. Œdema is marked, especially in the face (Fig. 83); the urine is scanty, and contains much blood, albumin and many casts. Very often some other syphilitic manifestation is also present. At post-mortem the kidneys are found to be filled with miliary gummata. *Paroxysmal hæmoglobinuria* is nearly always a syphilitic manifestation. Chills will bring on an exacerbation in such cases, the urine is passed a bright-red colour, and on inspection the red cells have all been laked, so that the hæmoglobin tints the urine a bright pink.

Fig. 83.—Case of syphilitic nephritis, aged six months, showing generalized œdema.

The teeth.—Peg-shaped or Hutchinsonian teeth (Fig. 84) are extremely characteristic of congenital syphilis. These appear in the second dentition only. The broadest portion of the peg is next the gum, the free edge being narrow in all dimensions. In the free edge is a characteristic crescentic notch. Carious teeth, however, often wear to such a shape and may be mistaken for syphilitic teeth.

Summary.—Congenital syphilis manifests itself, then, in infancy in a rash, snuffles, pseudo-paralysis, occasional nephritis, condylomata about the mouth and anus, and hydrocephalus. The *later manifestations* of syphilis are

interstitial keratitis, saddle-shaped nose, Hutchinsonian teeth, painless bilateral swellings of the knees (Fig. 85,) perforation of the palate, cirrhosis of the liver, sabre-shaped tibiæ, osteitis and periostitis of the various bones, bossing of the skull.

Pathology.—As each manifestation of syphilis has

Fig. 84.—Congenital syphilis, showing Hutchinsonian teeth.
(*By courtesy of Dr. Nabarro.*)

been dealt with a word has been said as to its pathology. It may be added, however, that the *Spirochæte pallida* causes an inflammation near a blood-vessel, the reaction to which shows the presence of round cells, and, later, fibrosis occurs. The spirochæte is best demonstrated in the liver, but we depend largely on the Wassermann reaction for the confirmation of the diagnosis.

Treatment.[1]—Just as in adult syphilis, the longer

[1] D. N. Nabarro, "The Treatment of Congenital Syphilis in Children," *Lancet*, June 5, 1926, pp. 1108–9.

Fig. 85. Case of congenital syphilis, aged 8, showing bilateral enlargement of the knees, bossed skull and depressed bridge to nose.

the lesion has existed the harder it is to eradicate the infection from the body, and the more likelihood there is of permanent damage. In congenital syphilis the infection was acquired in utero and had existed for some months before the child was born. The infection is therefore very widely spread throughout the tissues and some of the vital organs are already grossly damaged.

Of the **arsenic preparations** available the two most used in children are Novarsenobillon (N.A.B.) and Sulfarsenol. The dosage is as follows:—

Age.	*Initial dose of N.A.B.*	*Full dose of N.A.B.*	*Initial dose of Sulfarsenol.*
1 month	0.05 grm.	0.05—1 grm.	1½ cgm. to 8-12 cgm.
1-3 years	0.1 grm.	0.1 —0.25 grm.	——
Over 3 years	0.15 grm.	0.15—0.3 or 0.45 grm.	4 or 6 cgm. to 24 or 36 cgm.

Six injections are given at intervals, gradually increasing from the initial to the full dose. In the case of N.A.B. the dose of 0.45 grm. is not to be exceeded in a child under 12 years of age.

Technique of administration.—Sterilized distilled water, 1 to 2½ c.c., is used to dissolve the preparation. N.A.B. is best given intravenously or intramuscularly. Sulfarsenol may be given subcutaneously or intramuscularly. If either is administered directly into the muscle of the buttock the results are almost painless. Sulfarsenol is the least painful of the arsenic preparations when injected subcutaneously; it is best given beneath the abdominal wall. When the preparations are given intravenously a vein at the bend of the elbow or at the ankle should be chosen. No better syringe than the all-glass three-piece syringe sold by Burroughs, Wellcome and Co. is available. The longitudinal sinus should not be used for administering the arsenic preparations, but it may be utilized in taking the blood for the Wassermann test.

Treatment by mercury.—Hydrarg. c. creta may be given by the mouth in doses of $\frac{1}{4}$ grain to 2 or 3 grains, twice daily. In addition mercury ointment may be used, a small piece the size of a pea being rubbed into a different portion of the child's body each day. These forms of treatment should be continued until the clinical manifestations of the disease have cleared up, until there is no further recurrence of the disease, and finally until the blood Wassermann test is negative.

Children in whom the Wassermann reaction gives a positive result but who exhibit no symptoms or signs of the disease should be treated as a prophylactic measure. Again, children coming from a syphilitic family but who themselves have a negative Wassermann should also be treated, lest they develop active manifestations of syphilis later. Finally, a syphilitic mother who is pregnant should be treated during pregnancy in order that her infant may be born free from syphilis.

Prognosis.[1]—Infants in whom there are gross manifestations of syphilis at birth or shortly after birth show an extremely high mortality and, even if they survive, exhibit as a rule some permanent disability. If the manifestations are delayed for some weeks or months, and immediately they do appear treatment is begun, there is good hope of the child being quite cured, that is, the clinical manifestations clear up, and there is a permanently negative result to the Wassermann reaction, no further recurrences take place. Of all the manifestations, eye symptoms are the most difficult to eradicate. In the mentally defective child improvement takes place, but not recovery. It is obvious that the ultimate prognosis depends entirely on the organs affected and the duration of the disease affecting them.

[1] Donald Paterson, "Transmitted Congenital Syphilis," *Brit. Journ. Child. Dis.*, Oct.–Dec. 1920, Vol. 17, pp. 197–198.

APPENDIX

Quantities of certain drugs which may be given to children at different ages

These doses may be given three times daily by the mouth, unless otherwise stated.

Drug	*At birth*	*6 months*	*1 year*	*5 years*	*10 years*
Aloes				$\frac{1}{10}$ gr.	$\frac{1}{5}$ gr
Ammon. acetatis, liq.	3 min.	4 min.	5 min.	7 min.	15 min.
Ammon. aromat., spirit.	$\frac{1}{4}$ min.	$\frac{1}{2}$ min.	1 min.	2 min.	3 min.
Ammonium carbonate	$\frac{1}{8}$ gr.	$\frac{1}{4}$ gr.	$\frac{1}{2}$ gr.	2 gr.	3 gr.
Arsenicalis, liq.	$\frac{1}{4}$ min.	$\frac{1}{2}$ min.	1 min.	2 min.	3 min.
Aspirin	$\frac{1}{4}$ gr.	1 gr.	$1\frac{1}{2}$ gr.	$2\frac{1}{2}$ gr.	5 gr.
Atropine by the mouth	$\frac{1}{240}$ gr.	$\frac{1}{120}$ gr.	$\frac{1}{60}$ gr.	$\frac{1}{30}$ gr.	$\frac{1}{15}$ gr.
Atropine (hypod. injec.)	$\frac{1}{300}$ gr.	$\frac{1}{150}$ gr.	$\frac{1}{100}$ gr.	$\frac{1}{50}$ gr.	$\frac{1}{25}$ gr.
Belladonna, tinc.	2 min.	3 min.	4 min.	5 min.	10 min.
Bismuth carb.	3 gr.	5 gr.	10 gr.	20 gr.	30 gr.
Calcium lactate	1 gr.	$2\frac{1}{2}$ gr.	5 gr.	7 gr.	10 gr.
Calomel	$\frac{1}{10}$ gr.	$\frac{1}{8}$ gr.	$\frac{1}{4}$ gr.	$\frac{1}{2}$ gr.	$\frac{1}{2}$ gr.
Camph. co., tinc.	1 min.	2 min.	3 min.	5 min.	10 min.
Camphor (hypod. injec.)	$\frac{1}{10}$ gr.	$\frac{1}{8}$ gr.	$\frac{1}{4}$ gr.	$\frac{1}{2}$ gr.	$\frac{1}{2}$ gr.
Cascara, liq. ext.				30 min.	30 min.
Chloral hydrate	$\frac{1}{2}$ gr.	1 gr.	2 gr.	4 gr.	7 gr.
Chloretone (P. D. & Co.)			1 gr.	2 gr.	4 gr.
Ether, spirit of nitrous	$\frac{1}{4}$ min.	$\frac{1}{2}$ min.	1 min.	2 min.	3 min.
Ferri et ammon. cit.	$\frac{1}{2}$ gr.	$\frac{1}{2}$ gr.	1 gr.	3 gr.	5 gr.
Ferri iodidi, syr.	3 min.	5 min.	10 min.	30 min.	30 min.
Ferri phos. co., syr.	3 min.	5 min.	10 min.	30 min.	30 min.
Ferri et quinine cit.	$\frac{1}{4}$ gr.	$\frac{1}{2}$ gr.	1 gr.	3 gr.	5 gr.
Hexamine	$\frac{1}{2}$ gr.	1 gr.	2 gr.	5 gr.	5 gr.
Hydrarg. c. creta	$\frac{1}{4}$ gr.	$\frac{1}{2}$ gr.	1 gr.	5 gr.	7 gr.
Ipecac., vinum	1 min.	2 min.	3 min.	5 min.	5 min.
Liquorice, liq. ext.				30 min.	30 min.
Nux vom., tinc.	$\frac{1}{2}$ min.	1 min.	2 min.	4 min.	6 min.
Phenolphthalein	$\frac{1}{6}$ gr.	$\frac{1}{4}$ gr.	$\frac{1}{2}$ gr.	1 gr.	1 gr.
Phenazone	$\frac{1}{10}$ gr.	$\frac{1}{4}$ gr.	$\frac{1}{2}$ gr.	2 gr.	4 gr.
Pot. bromide	1 gr.	2 gr.	4 gr.	6 gr.	10 gr.
Pot. cit.	$\frac{1}{2}$ gr.	1 gr.	3 gr.	5 gr.	10 gr.
Pot. iod.	$\frac{1}{4}$ gr.	$\frac{1}{2}$ gr.	1 gr.	2 gr.	3 gr.
Quinine sulphate	$\frac{1}{10}$ gr.	$\frac{1}{8}$ gr.	$\frac{1}{4}$ gr.	$\frac{1}{2}$ gr.	1 gr.
Rhei, pulv.	$\frac{1}{4}$ gr.	$\frac{1}{2}$ gr.	1 gr.	$2\frac{1}{2}$ gr.	5 gr.
Santonin				2 gr.	3 gr.
Scillæ oxymel	1 min.	2 min.	3 min.	5 min.	7 min.
Senna, syr.	2 min.	4 min.	6 min.	10 min.	16 min.
Sod. bicarb.	2 gr.	3 gr.	5 gr.	10 gr.	20 gr.
Sod. brom.	1 gr.	2 gr.	4 gr.	6 gr.	10 gr.
Sod. cit.	$\frac{1}{2}$ gr.	1 gr.	3 gr.	5 gr.	10 gr.
Sod. sal.	$\frac{1}{2}$ gr.	1 gr.	2 gr.	3 gr.	5 gr.
Stramonium, tinc.	$\frac{1}{2}$ min.	1 min.	2 min.	3 min.	5 min.
Strychnine (hypod. injec.)	$\frac{1}{1000}$ gr.	$\frac{1}{800}$ gr.	$\frac{1}{600}$ gr.	$\frac{1}{200}$ gr.	$\frac{1}{100}$ gr.

INDEX

A

Abdomen, in normal child, 13
—— inspection of, 13
—— palpation of, 13
—— unduly large, significance of, 13
Abdominal glands, inflamed, simulating appendicitis, 175
—— —— tuberculosis of (*see* Tuberculosis, abdominal)
—— pain due to tuberculosis, 428
—— —— in appendicitis, 173, 175, 176
—— —— in Henoch's purpura, 248
—— tuberculosis (*see* Tuberculosis, abdominal)
Abscess, appendicular, 430
—— cerebral(*see* Cerebral abscess)
—— of breast preventing breast-feeding, 74
—— peritonsillar, 151
—— retropharyngeal, 151
—— tuberculous cervical, 433
"Absent moment," in epilepsy, 340
Acarus, 375
Accidents and violence, mortality due to, 33, 34
Acetonæmia as cause of vomiting, 358
Acetone bodies and acidosis, 132
—— odour of,in breath in diabetes, 463
Acholuric family jaundice (*see* Jaundice, acholuric family)
Achondroplasia, 366
—— arms and legs in, 13
Acid dermatitis, 373
Acidosis, 132
—— and appendicitis, 175
—— and enuresis, 263
—— and mucous colitis, 178
Acidosis as cause of cyclical vomiting, 132
—— —— of urticaria, 382
—— chronic, 128
—— complicating nose and throat infections, 151
—— due to insufficient sugar, 82
—— fat dyspepsia and, 121
—— in diabetes, 469
—— in nervous exhaustion, 25, 353
—— things to be avoided in, 135
—— (*see also* Vomiting, cyclical)
Acidosis-producing diet in treatment of epilepsy, 343
Adenitis, inguinal and mesenteric, diagnosis of appendicitis from, 174
—— tuberculous cervical (*see* Tuberculous cervical adenitis)
Adenoids as cause of chest deformity, 12
—— —— of disturbed sleep, 27
—— —— of night terrors, 357
—— infected, as cause of vomiting, 358
—— —— removal of, in nasal diphtheria, 405
—— nose in, 9
—— preventing breast-feeding, 74
Adenoma of thyroid, 338
Adherent pericardium, 227, 228, 454
Adiposity (*see* Obesity)
Adoption of children, 32
Aerophagia, 142
—— breaking of wind in, 142, 143
—— causes of, 143
—— test-feeds in, 143
—— treatment of, 143
Agarol, 139
Agglutination test (*see* Widal test)
Air-hunger, 125, 133, 464

Albulactin, 80
Albumen-water to be avoided in dietetic diarrhœa, 122
Albumin in cow's milk, 62, 70
—— in human milk, 62, 70
—— serum, 254
Albuminuria, 253
—— orthostatic, 252, 253
—— —— treatment of, 254
—— postural, 253
Alcohol injections in prolapse of rectum, 187
—— instillation of, in otitis media, 148
Alimentary tract, tuberculosis of, 418
—— (*see also* Tuberculosis, abdominal)
Alkalis in treatment of pyelitis, 259
—— use of to modify milk, 81
Allenburys' dried milk, 67, 85, 88
—— feeding-bottle, 88
—— Malted Food No. 3, 67, 90
Allergy and asthma, 220
Almata dried milk, 67, 387
Alopecia, 378
Amaurotic family idiocy, 48
Ambrosia full-cream dried milk, 66, 85
—— humanized dried milk, 67
Amentia, congenital, 322, 326
Ammonia dermatitis, 373
Ammoniacal odour on waking child, 374
Amyotonia congenita, 309
Anæmia, 233
—— accompanied by cardiac dilatation, 226, 227
—— in acute lymphatic leukæmia, 240
—— in chronic parenchymatous nephritis, 278
—— in lymphadenoma, 243
—— in rheumatism, 449, 458
—— in spleno-medullary leukæmia, 241
—— of mother a contra-indication to breast-feeding, 71
—— of premature infants, 61
—— splenic, 233
—— —— age-incidence of, 185
—— —— hæmatemeses in, 233
Anæmia, splenic, infantile (*see* Anæmia, von Jaksch's)
—— —— jaundice in, 233
—— —— liver in, 233
—— —— spleen in, 233
—— von Jaksch's, 236
—— —— blood-picture in, 238
—— —— diet in, 238
—— —— etiology and pathology of, 236
—— —— prognosis of, 238
—— —— symptoms and diagnosis of, 238
—— —— treatment of, 238
—— (*see also* Chlorosis)
Anæsthesia in tonsillectomy, 153
—— in treatment of pyloric stenosis, 167
Anal fissure, 138
—— —— as cause of constipation in infants, 136, 138
—— stenosis as cause of constipation in infants, 136
Anaphylaxis as factor in asthma, 219, 220, 222
—— in rheumatoid arthritis, 461
—— theory of eczema, 384
Angier's Emulsion, 137
Anginal attacks in congenital heart disease, 232
Ankle, tuberculosis of, 437
Anorexia, causes of, 355
—— diet and, 356
—— in premature infants, 59
—— nervosa, 356
—— treatment of, 356
Anterior poliomyelitis (*see* Poliomyelitis)
Anti-acidosis diet, 134
Antidiphtheritic serum in diphtheritic croup, 193
—— —— rash, 384
Antineuritic vitamin, 105
Antipellagra vitamin, 105
Antirachitic measures, 104
—— —— in treatment of flat-foot and knock-knee, 362
—— vitamin, 106
Anti-scarlet-fever serum, 393
Antiscorbutic vitamin, 105
Antiscorbutics, 109
Antistreptococcal serum, 36, 37, 393

Antistreptococcal serum rash, 384
Antitetanic serum, 38
—— —— rash, 384
Antitoxin serum in diphtheria, 404, 405
—— —— streptococcal, in pericarditis, 454
—— treatment in post-diphtheritic paralysis, 314
Antrum, disease of, complicating nose and throat infections, 152
—— —— tonsillar infections and, 152
Anuria complicating parenchymatous nephritis, 276
Aphthous stomatitis, 156
Aplasia, congenital cerebral, 41
Appendicectomy, when it is advisable, 176
Appendicitis, 172
—— abscess in, 172
—— acute, simulated by Henoch's purpura, 247
—— chronic, 174
—— —— differential diagnosis of, 175
—— —— symptoms and clinical picture of, 175
—— —— X-ray diagnosis of, 175
—— differential diagnosis of, 173
—— epidemic encephalitis mistaken for, 285
—— etiology and pathology of, 172
—— gangrene in, 172
—— mortality of, 33, 34
—— prognosis of, 174
—— rectal examination in, 174
—— recurrent, 174
—— rheumatism and, 172
—— subacute, 175
—— symptoms and diagnosis of, 173
—— treatment of, 174
—— with retro-cæcal position, 173
Appendicular abscess, differentiated from tuberculous peritonitis, 430
Appetite, lack of (*see* Anorexia)
Armour's peptone in asthma, 224
Arms, ataxia of, test for, 14
—— in achondroplasia, 13
Arrowroot, 68
Arsenic preparations in syphilis, 486
Arsenic-and-ipecacuanha mixture, 157
Arthritis, leading to septic meningitis, 298
—— pneumococcal, complicating lobar pneumonia, 209
—— rheumatic, 441, 448, 450
—— rheumatoid (*see* Rheumatoid arthritis)
—— syphilitic, 460
Artificial feeding (*see* Feeding, artificial)
—— sunlight (*see* Sunlight, artificial)
Ascaris lumbricoides, 472, 475
Aschoff's nodules, 444
Ascites, cardiac and renal, differentiated from tuberculous peritonitis, 430
Asphyxia at birth, 42
—— due to enlarged tonsils, 28
Aspiration of pus in empyema, 213
Asthenia and cyclical vomiting, 133
Asthma, 219
—— age- and sex-incidence of, 219
—— allergy and, 220
—— anaphylaxis as factor in, 219, 220, 222
—— and eczema, 219, 221
—— and hay fever, 219
—— and urticaria, 219, 221
—— bronchial, 196, 221
—— chest in, 12, 221
—— classification of cases of, 221
—— climate and, 219
—— clinical picture in, 220
—— diagnosis of, 220, 221
—— diet in, 221, 223
—— etiology of, 219
—— exciting factors in, 151, 219
—— heredity and, 219
—— leading to bronchitis, 220, 221
—— lips in, 10
—— medicines, 222
—— missing factor in, 220
—— nose in, 9
—— physical signs in, 221
—— prognosis of, 224

Asthma, pulmonary tuberculosis and, 224
—— recurrence of, 224
—— spasmodic, 221
—— thymic, 218
—— treatment of, after skin-tests, 224
—— —— of attack of, 222
—— —— dietetic régime in, 221
—— —— drugs employed in, 222
—— —— peptone injections in, 224
—— —— preliminary investigations in, 223
—— —— preventive, 221
—— —— skin protein tests in, 223
—— —— tuberculin injections in, 224
Ataxia of arm, 14
Atelectasis of lung, congenital, 52
—— —— —— as cause of retarded growth, 26
—— —— —— clinical picture in, 52
—— —— —— etiology and pathology of, 52
—— —— —— in premature infants, 60
—— —— —— preventing breast-feeding, 74
—— —— —— treatment of, 53
Atonicity of bowel, as cause of constipation, 138
Atresia, duodenal, 169
Atrophy, progressive spinal muscular (*see* Werdnig-Hoffmann paralysis)
—— yellow, acute or subacute, 184
—— —— catarrhal jaundice and, 183

B

Babinski's reflex in infants, 14
Bacillus typhosus, 405
—— paratyphosus-A and -B, 410
Balanitis, 269
Bananas in treatment of cœliac disease, 114
Banti's disease (*see* Anæmia, splenic)
Barlow's disease, 106
Basal murmurs usually congenital, 231
Beading of ribs in rickets, 12, 100, 103
—— —— in scurvy, 108
Bed-wetting (*see* Enuresis)
Belladonna rash, 384
" Bellows " murmur in rheumatic carditis, 228, 453
Bemax, 105, 139
Benger's Food, 89, 123, 125, 142, 180, 205, 272, 387
Benzoin mixture, compound tincture of (Hospital for Sick Children), 217
Beriberi, 105
Berina Food, composition of, 67
Berna condensed milk, composition of, 65
Bile-ducts, congenital absence of, 38, 50, 51, 185
Bilious attack, appendicitis mistaken for, 173
—— attacks, asthma and, 219, 221
Binder, 17
Birth injury, 41, 322
—— —— acquired mental defect and, 322
—— —— as cause of squint, 144
—— —— asymmetry of head due to, 8
—— —— convulsions due to, 48, 339, 348
Bismuth mixtures in dietetic diarrhœa, 124
Bladder, control of, defective treatment of, 16
—— —— development of, 15
Bleeders (*see* Hæmophilia)
" Bleeding time " lengthened in premature infants, 54
Blepharitis, chronic, 145
—— —— " exudative diathesis " and, 145
Blindness, 9
Blood, clotting of, delayed in premature infants, 42
—— diseases, 233
—— —— accompanied by cancrum oris, 158
—— —— lips in, 10
—— —— spleen in, 13
—— in stools, 97
—— —— (*see also* Melæna)

Blood in urine, 252
—— —— (*see also* Hæmaturia)
—— transfusion in acute lymphatic leukæmia, 241
—— —— in dietetic diarrhœa, 124
—— —— in empyema, 214
—— —— in hæmophilia, 250
—— —— in lymphadenoma, 247
—— —— in pneumococcal peritonitis, 177
—— —— in pyloric stenosis, 167
—— —— in spleno-medullary leukæmia, 242
—— —— in umbilical sepsis, 36, 37
—— —— in von Jaksch's anæmia, 238
Blood-pressure, normal, 251
Blood-sugar curve of case of diabetes, 463
Blood-urea, high in interstitial nephritis, 253
—— normal value of, 253
—— test, 253
Blue Butterfly condensed milk, composition of, 65
Boils (*see* Furunculosis)
Bone and vegetable broth, preparation of, 92
—— —— —— uses of, 61, 79, 90, 91
Bones and joints, diseases of, 362
—— syphilis of, 480
—— tuberculosis of, 418, 436
Bonnets for infants, 17
Bony deformity (*see* Dwarfism, renal)
—— rickets, 102
Bordet bacillus as cause of whooping-cough, 398
Bottle, feeding, 88
—— —— Allenburys', 88
—— —— boat-shaped, 88
—— —— Glaxo, 88
—— —— Soxhlet, 88
—— —— teat of, too small, causing wind-swallowing, 143
—— —— upright, 88
Bow-legs, 13
Bowel, emptying of, training in, 15, 16
—— perforation of, in typhoid fever, 408, 410
Bowel, tuberculous ulceration of, 426, 427, 428, 429, 431
Bowel-wall, inflammation of, 97
Bowman's mixture, 157
Brain lesions, causing squint, 144
—— —— inflammatory, epilepsy due to, 339
—— —— nystagmoid movements due to, 9
—— malformations, epilepsy due to, 339
—— tumour, causing drowsiness, 28
—— —— differentiated from epidemic encephalitis, 287
—— —— —— from tuberculous meningitis, 291
—— —— simulating epilepsy, 342
—— —— squint due to, 9
—— water on (*see* Hydrocephalus)
—— (*see also* Cerebellar; Cerebral)
"Bread-and-butter" children, 131
Breaking of wind, 73, 142, 143
Breast douching, 75
—— infections preventing breast-feeding, 74
—— massage, 75
Breast-fed infants, mixed feeding for, 89
Breast-feeding (*see* Feeding, breast-)
Breast-milk (*see* Milk, breast-)
Breasts, antenatal treatment of, 69
Breath-holding convulsions, 349
Breath-sounds, in normal child, 12
Breck feeder, 59
"Brewer's drayman" type of gigantism, 27
Brittle bones and blue sclerotics, 367
Bromide rash, 383
Bromides, in treatment of epilepsy, 344
Bronchial asthma, 196, 221
Bronchiectasis, 214
—— and tuberculosis, 216
—— auscultation in, 216
—— cardiac failure in, 217
—— cerebral abscess in, 217
—— complicating broncho-pneumonia, 202
—— —— lobar pneumonia, 210
—— cough in, 215

Bronchiectasis, diagnosis of, 214
—— —— from pulmonary tuberculosis, 422
—— etiology of, 214, 400
—— examination of chest in, 215
—— hæmoptysis in, 217
—— pathology of, 214
—— percussion of chest in, 216
—— prognosis of, 217
—— simulating asthma, 221
—— sputum in, 215
—— symptoms and diagnosis of, 214
—— treatment of, 217
—— whooping in, 215
—— X-ray examination in, 216
Bronchiolectasis (*see* Bronchiectasis)
Bronchitis, 151, 194
—— and asthma, 220, 221
—— as cause of anorexia, 355
—— clinical picture in, 196
—— cough in, 196
—— etiology of, 194
—— mortality of, 33
—— pathology of, 195
—— physical signs of, 195
—— temperature in, 196
—— treatment of, 196
Broncho-pneumonia (*see* Pneumonia, broncho-)
Bronchus, foreign bodies in, causing bronchiectasis, 214
—— —— body in, simulating mediastinal gland tuberculosis, 422
Bulbar paralysis due to polioencephalitis, 307
Bullous impetigo, 39
Burney Yeo mask, 217
Bus sickness, 219
Butolan, 477
Buttermilk, 87
Buttocks, "burning" of, 97
—— excoriation of (*see* Dermatitis)
Butyric acid in stools, 373
Bynol, 357

C

Calcification, defective, causes of, 30, 106
Calcium deficiency and dental caries, 160
—— —— and rickets, 102, 103
—— —— condensed milk and, 103
—— —— in premature infants, 54, 61
—— salts in treatment of hæmophilia, 250
—— therapy for chilblains, 375
Calculus, 254
—— causing frequency of micturition, 266
—— simulating appendicitis, 175
Callard's obesity bread, 21
Caloric requirements of premature infants, 55
—— value of dried milks, 66, 67
—— —— of starchy patent foods, 67
Cancrum oris, 158
Caps, for infants and children, 17
Caput succedaneum, 8, 45
Carbohydrate dyspepsia, 119, 120, 125
—— excess of in diet, causing acid dermatitis, 373
—— of cow's milk, 82
—— percentage of in dried milks, 66, 67
—— —— in starchy patent foods, 67
Cardiac (*see* Heart)
Cardio-respiratory murmur, significance of, 227
Cardiospasm, 161
Cardio-vascular instability and cyclical vomiting, 133
Carditis, 227, 448, 452
—— rheumatic (*see* Rheumatic carditis)
—— syphilitic, 482
Caries, dental (*see* Dental caries)
—— spinal (*see* Spinal caries)
Carnation condensed milk, composition of, 65
Carpopedal spasm in rickets, 101
Casec dried milk, 66, 80
Casein in milk, breast-, 62, 70
—— —— cow's, 62, 70, 81
Castor oil causing catarrh of bowel, 179
Catarrh, 151, 195
Catarrhal jaundice (*see* Jaundice, catarrhal)

Catarrhal mucous colitis, 179
Catatonia, in epidemic encephalitis, 284
Catheter, Eustachian, 149, 258
Catheterization of female infants, 251, 258
Cellulitis of deep tissues, 371
Cephalhæmatoma, 45
—— clinical picture in, 45
—— treatment of, 46
Cephalic cry, in tuberculous meningitis, 290
Cereal, cooking of, 90
—— waters for dilution of milk, 81
Cereals, in constipation, 137
Cerebellar tumour, gait due to, 14
—— —— tilting of head due to, 11
Cerebral abscess, differentiated from tuberculous meningitis, 291
—— —— following bronchiectasis, 298
—— —— following otitis media, 298
—— —— in bronchiectasis, 217
—— —— leading to septic meningitis, 298
—— aplasia, congenital, 41
—— blindness, 9
—— damage at birth, predisposing to convulsions, 348
—— degeneration, 48, 342
—— hæmorrhage, accompanying interstitial nephritis, 280
—— —— as cause of convulsions, 342
—— —— of epilepsy, 339
—— —— of squint, 144
—— —— at birth, 41, 42
—— thrombosis, complicating lobar pneumonia, 210
—— tumour as cause of epilepsy, 339
—— —— —— of facial paralysis, 315
Cerebro-spinal fluid in acute anterior poliomyelitis, 304
—— —— in epidemic encephalitis, 285, 295
—— —— in meningitis, 291, 295, 299, 300
Cerebro-spinal fluid in poliomyelitis, 295, 304
—— meningitis (*see* Meningitis, cerebro-spinal)
Certified milk, 62
Cervical adenitis, complicating scarlet fever, 392
—— —— tuberculous (*see* Tuberculous cervical adenitis)
—— glands (*see* Glands, cervical)
Chapman's Whole Wheat Flour, composition of, 68
Chest, auscultation of, 12
—— —— abnormalities in, significance of, 12
—— barrel-shaped, 12
—— deformities in rickets, 99
—— examination of, 11
—— palpation of, 11
—— percussion of, 12
—— pigeon-, 12
—— shape of, 11
Chicken-pox, 402
—— and herpes zoster, 380
—— and urticaria, 383
—— complications of, 402
—— etiology of, 402
—— incubation period of, 402
—— quarantine period and rules to be observed in, 390
—— rash of, 402
—— return to school after exposure to infection of, 390
—— symptoms and clinical picture of, 402
—— treatment of, 402
Chilblains, 374
—— treatment of, 374
Child, normal, examination of, 7
—— premature (*see* Premature infant)
Childhood, mortality in, from various causes, 33, 34
Children, adoption of, 32
"Chill," 195
"Chilprufe" clothing, 17
Chlorosis, 239
Chorea, 444
—— as cause of anorexia, 355
—— cardiac accompaniments of, 446
—— clinical picture in, 445
—— etiology of, 444

Chorea, involvement of muscles in, 445
—— knee-jerks in, 446
—— pathology of, 445
—— prognosis of, 446
—— pulse-rate in, importance of, 447
—— sex-incidence of, 441, 445
—— treatment of, 447
Choroiditis, syphilitic, 482
Chvostek's sign in rickets, 10, 101
—— —— in spasmophilia, 350
Circulatory system, diseases of, 225
Circumcision, 268, 359
"Circumoral pallor" in scarlet fever, 392
Cirrhosis of liver (*see* Liver, cirrhosis of)
Cistern puncture, 320
—— —— in meningitis, 296, 299, 300
—— —— preparation of patient in, 320
—— —— technique of, 321
Cleft palate, 50
—— —— and hare-lip preventing breast-feeding, 74
Climate and asthma, 219
Clothes of infant, shortening of, 16
Clothing of infants and children, 16
Club-foot, 50
Clubbing of fingers in bronchiectasis, 215
—— —— in congenital heart disease, 229, 231
Cod-liver oil, 64, 90, 91
—— —— as cause of urticaria, 383
—— —— as source of vitamin A, 105
—— —— —— of vitamin D, 106
—— —— in abdominal tuberculosis, 432
—— —— in anorexia, 355
—— —— in chilblains, 375
—— —— in cœliac disease, 114
—— —— in cretinism, 338
—— —— in dentition, 30, 31
—— —— in empyema, 213
—— —— in flat-foot and knock-knee, 362
—— —— in nephritis, 277, 282
Cod-liver oil in phlyctenular conjunctivitis, 145
—— —— in rickets, 104
—— —— in spasmophilia, 350
—— —— in treatment of premature infant, 61
—— —— in von Jaksch's anæmia, 238
—— —— in whooping-cough, 401
Cœliac disease, 110
—— —— abdomen in, 13, 110
—— —— adult sprue and, 113
—— —— age-incidence of, 110
—— —— and prolapse of rectum, 186, 187
—— —— and tendency to umbilical hernia, 190
—— —— diet in, 115, 431
—— —— etiology and pathology of, 110
—— —— infantilism in, 26, 110, 114
—— —— liver in, 13, 110, 113
—— —— mortality of, 113
—— —— prognosis of, 113
—— —— sex-incidence of, 110
—— —— stools in, 110, 113
—— —— symptoms and diagnosis of, 110
—— —— treatment of, 114
—— —— tuberculous peritonitis and, 110
Cold, common, 150, 381
—— weather, breast-milk requirements in, 77
Colic as cause of disturbed sleep, 357
—— intussusception, 180
—— mistaken for appendicitis, 176
—— umbilical (*see* Umbilical colic)
—— wind, 28, 76
Colitis, 178
—— due to worms, 473, 476
—— mucous, 178
—— —— clinical picture of, 179
—— —— diet in, 179
—— —— etiology of, 178
—— —— stools in, 179
—— —— treatment of, 179
Colonic lavage in pyloric stenosis, 168, 169
Colostrum, 69

Complementary feeding, 71, 72, 74, 77
—— —— rules for, 77
Complexion of normal child, 10
Concomitant squint, 144
Condensed milk (*see* Milk, condensed)
Condylomata, syphilitic, 372, 479, 480
Congenital absence of bile-ducts, 38, 50, 51, 185
—— —— of thyroid, 26
—— anal stenosis, 136
—— atelectasis of lung, 26, 52, 60, 74
—— cerebral aplasia, 41
—— cretinism, 333
—— cystic kidney, 26, 255
—— dislocation of hip, 13, 50, 362
—— heart disease (*see* Heart disease, congenital)
—— hydrocephalus, 322, 327
—— malformations, 12, 49
—— mental defect, 322, 323
—— morbus cordis, 229
—— pyloric stenosis (*see* Pyloric stenosis, congenital)
—— syphilis (*see* Syphilis, congenital)
Conjunctivitis, 144
—— accompanying rubella, 394
—— complicating measles, 395
—— due to Koch-Weeks bacillus, 144
—— phlyctenular, 145
—— —— tuberculin test in, 439
—— treatment of, 144, 145
Constipation, 135
—— and colic mistaken for appendicitis, 176
—— complicating pneumonia, 202
—— due to congenital pyloric stenosis, 164
—— in artificially-fed infants, 136
—— in breast-fed infants, 75, 83, 135
—— in congenital heart disease, 232
—— in nursing mother, 73
—— in older children, 137
—— in tuberculous meningitis, 290
—— in underfeeding (*see* Underfeeding)
Constipation, treatment of, 136, 137, 138, 139
—— —— prescriptions for, 140
Contacts, protection of, in scarlet fever, 393
—— quarantine for, 389, 390
Convergent squint, 144
Convulsions, 48, 339, 347
—— complicating pneumonia, 202, 208
—— due to birth injury, 48, 339, 348
—— due to breath-holding, 349
—— due to cerebral hæmorrhage, 42, 48
—— due to cerebral nævi, 48
—— due to cerebral tumour, 48
—— due to congenital brain malformations, 48, 348
—— due to epilepsy, 48, 339, 346
—— due to hyperpyrexia, 350
—— due to meningitis, 48
—— due to rickets, 48, 102, 347, 350
—— due to sepsis neonatorum, 48
—— due to spasmophilia, 48, 102, 350
—— due to tetany, 48, 102
—— due to whooping-cough, 349, 400
—— in epilepsy, 339, 346
—— in hydrocephalus, 331
—— in infants, 347
—— —— age and, 348
—— —— dehydration and, 349
—— —— diagnosis of, 347
—— —— during teething, 31
—— —— infections and, 348
—— —— new-born, 48
—— —— predisposing causes of, 347
—— —— prognosis of, 351
—— —— stages of, 347
—— —— treatment of, 351
—— —— typical signs of, 347
—— —— uræmic, 261, 351
—— in tuberculous meningitis, 291
—— with fever, 342, 348
Cornea, ulceration of, 145, 380
Corneal opacities due to syphilis, 482
Coronet condensed milk, composition of, 65

Cough during dentition, 28, 31
—— in bronchitis, 196
—— in pulmonary tuberculosis, 421
—— mixtures, 197
Cow and Gate dried milk, 85, 180
—— —— —— —— full-cream, composition of, 66
—— —— —— —— half-cream, composition of, 67
—— —— —— —— mixture, 85
—— —— —— —— skimmed, composition of, 66
Cracked nipples, 74
"Cramps," due to rickets, 102
Craniotabes, 101
Cream of Rice (Groult's), composition of, 68
—— of Wheat, 68, 91, 134, 142, 276
Cresolene lamp in bronchitis, 197
—— —— in croup, 192
Cretinism, 322, 333
—— acquired, 23, 338
—— and mongolism, differences between, 335
—— as cause of retarded growth, 26
—— congenital, clinical picture in, 334
—— —— etiology and pathology of, 333
—— —— prognosis of, 336
—— —— thyroid gland in, 334
—— —— tongue in, 334
—— —— treatment of, 336
—— —— —— danger of rickets following, 338
—— —— —— dosage of thyroid in, 336, 337
—— —— —— metabolic estimations in, 337
—— —— —— signs of excessive thyroid in, 337
—— —— —— —— of recovery in, 336
—— mouth in, 10
Cretinism, umbilical hernia in, 190
Croup, diphtheritic, 191, 193
—— spasmodic, 191
—— —— age-incidence of, 191
—— —— clinical picture in, 191
—— —— differential diagnosis of, 192
—— —— prognosis in, 192
—— —— treatment of, 192
Crowing, in rickets, 102
Curd of cow's milk (*see* Milk, cow's, curd of)
Curds, in stools, 82, 97
Cyanosis in congenital heart disease, 231
—— in premature infants, 60
Cyclical vomiting (*see* Vomiting, cyclical)
Cystic kidney, congenital, 26, 255
Cystitis, 262
—— causing frequency of micturition, 266
—— —— hæmaturia, 254
—— due to administration of hexamine, 306

D

Dactylitis, syphilitic, 481
Dampness and rheumatism, 442, 458
De Pezzer drainage tube, 213
Deafness, tests for, 9
Death, causes of (*see* Mortality)
Debility and fat dyspepsia, 129
—— as cause of constipation, 138
Defæcation, training of child in good habits of, 15, 16
Deficiency diseases, 105
Deformity of bones (*see* Dwarfism, renal)
Dehydration as cause of convulsions, 349
—— "blue turns" due to, 60
—— fever, 83, 127
—— in dietetic diarrhœa, 120
—— —— —— treatment of, by saline, 123
—— in infective diarrhœa, 119
Demerara sugar, 83, 85
Dental caries, 159
—— —— and enlarged cervical glands, 160
—— —— and nervousness, 160

Dental caries and septic tonsils, 160
—— —— causes of, 160
—— —— evils of, 159
—— —— malnutrition and, 159
—— —— pathology of, 160
—— —— prevention of, 161
Dentition, 28, 29
—— breath-sounds in, 12
—— delayed, 29
—— imperfect, 29
—— primary, 29
—— secondary, 32
—— symptoms of, 30
—— theories of, 30
—— treatment during, 31
Dermatitis, acid, 373
—— ammonia, 373
—— exfoliative, 37
—— treatment of, 373
D'Espine's sign, 421
Development, normal and abnormal, 7
—— of special senses, 14
—— of speech, 14
—— of motor control, 14
Dextrimaltose, 82, 83, 85
Diabetes insipidus and enuresis, 262
—— mellitus, 462
—— —— and enuresis, 262
—— —— blood-sugar in, 463
—— —— clinical picture in, 462
—— —— coma of, 464
—— —— diagnosis of, 462
—— —— etiology and pathology of, 462
—— —— food requirements in, 466
—— —— hypoglycæmia in, 464, 470
—— —— insulin in, 462, 464, 465, 468
—— —— management of case of, 468
—— —— prognosis of, 464
—— —— renal glycosuria and, 464
—— —— treatment of, 465
—— —— —— common errors in, 468
—— —— —— diet in, 466, 469, 470
—— —— —— effect of aperients in, 469
Diabetes mellitus, treatment of, effect of exercise in, 469
—— —— —— food-tables for, 466, 470
—— —— —— general management of child in, 465, 468
—— —— —— influence of infections in, 469
—— —— —— menu for, 470
—— —— —— of hypoglycæmia in, 471
—— —— —— testing of urine in, 467
—— —— —— typical record of, 468
—— —— —— variations in composition of foodstuffs in, 469
—— —— weight-chart of case of, 465
Diarrhœa, accompanying eczema, 386
—— —— pneumonia, 202
—— —— septicæmia, 37
—— acute or chronic, leading to broncho-pneumonia, 200
—— chronic in tuberculous ulceration of bowel, 429
—— complicating empyema, 214
—— dietetic, 116, 119
—— —— acidosis in, 121
—— —— carbohydrate dyspepsia and, 120, 125
—— —— clinical picture in, 120
—— —— diagnosis of, 122
—— —— etiology and pathology of, 119
—— —— fat dyspepsia and, 119, 125
—— —— feeding in, 84, 122, 125
—— —— lactic acid milk in, 122, 125
—— —— prognosis of, 122
—— —— stools in, 120, 121, 122
—— —— temperature in, 120, 121
—— —— treatment of, 122
—— —— —— by saline, 123
—— —— —— by stimulants, 125
—— —— —— in severe cases, 125
—— —— —— medicinal, 124
—— due to pneumococcal peritonitis, 177

Diarrhœa during dentition, 30
—— epidemic, 116
—— fermentative, 125
—— in cœliac disease, 110
—— in ulcerative stomatitis, 157
—— infective, 116, 117
—— —— clinical picture in, 118
—— —— dehydration in, 119
—— —— etiology and pathology of, 117
—— —— prognosis of, 118
—— —— seasonal incidence of, 117
—— —— stools in, 118
—— —— temperature in, 117
—— —— treatment of, 119
—— summer, 116
—— symptomatic, 116, 126
—— —— accompanying broncho-pneumonia, 202
—— —— infections causing, 126
—— —— treatment of, 126
Diarrhœal diseases, mortality of, 35
Dick test, 393
Diet, acidosis-producing, in epilepsy, 343
—— and dermatitis, 373
—— anti-acidosis, 133, 134, 158
—— —— in ulcerative stomatitis, 158
—— anti-ketogenic, 133, 134, 158
—— badly balanced, causing acidosis, 132
—— Epstein's, 279
—— fever, 197, 415
—— for children aged from 1 to 2 years, 92
—— —— —— from 2 to 5 years, 93
—— —— of school age, 94
—— in abdominal tuberculosis, 431
—— in acute hæmorrhagic nephritis, 272
—— in asthma, 221, 223
—— in bronchitis, 197
—— in catarrhal jaundice, 183
—— in chorea, 447
—— in cœliac disease, 114, 115
—— in diabetes, 466, 470
—— in eczema, 387
—— in enuresis, 264
—— in epidemic encephalitis, 288
Diet in epilepsy, 343
—— in fat dyspepsia, 129
—— in infectious fevers, 415
—— in interstitial nephritis, 282
—— in mastoid disease, 415
—— in mucous colitis, 179
—— in obesity, 20
—— in otitis media, 415
—— in pink disease, 312
—— in pneumonia, 205, 415
—— in post-diphtheritic paralysis, 314
—— in rickets, 104
—— in scurvy, 109
—— in starch dyspepsia, 131
—— in typhoid fever, 408
—— in von Jaksch's anæmia, 238
—— in whooping-cough, 401
—— ketogenic, in epilepsy, 343
—— of nursing mother, 73
—— rachitic, as cause of dental caries, 160
—— (*see also* Feeding, mixed)
Dietetic diarrhœa (*see* Diarrhœa, dietetic)
Digestion, disturbances of, 116
—— —— as cause of night terrors, 358
—— —— as exciting factor in asthma, 219
Digestive system, diseases of, 155
Dilution of milk, 70, 81
Diphtheria, 403
—— age and, 403
—— albuminuria in, 253
—— clinical picture in, 403
—— complications of, 404
—— diagnosis of, 403
—— etiology of, 403
—— immunity in, 405
—— immunization in, 405
—— —— contra-indications to, 405
—— incubation period of, 403
—— leading to interstitial nephritis, 279
—— milk and, 63
—— mortality of, 33, 34, 404
—— nasal, 404
—— otitis media in, 146
—— paralysis, 14, 312
—— prognosis of, 404
—— Schick test in, 405

Diphtheria : susceptibles, treatment of, 405
—— symptoms of, 403
—— toxin, injection of, in Schick test, 405
—— treatment of, 404
Diploma condensed milk, composition of, 65
Dislocation of epiphysis, resemblance of to scurvy, 109
—— of hip (*see* Hip, dislocation of)
Disturbed sleep, causes of, 27, 357
Diuretin, 277
Diurnal somnolence, 28
Divergent squint, 144
Dorsella dried milk, 66, 85
Douching of breasts, 75
Dried milk (*see* Milk, dried)
Drinks, restriction of, in epilepsy, 344
Drooling in dentition, 28
Drug rashes, 383
Drugs, dosage of, for various ages, 488
Dryco dried milk, 66, 122
Duodenal atresia, 169
—— stenosis, 169
—— —— prognosis of, 170
—— —— pyloric stenosis and, 169
—— —— symptoms of, 169
—— —— treatment of, 170
—— ulcer, 171
—— —— gastro-enterostomy in, 172
—— —— melæna in, 171
—— —— treatment of, 172
Dwarfing due to rickets, 104
Dwarfism, 26
—— renal, 27, 279, 280, 281
Dysentery as cause of diarrhœa, 116
Dyspepsia as cause of disturbed sleep, 358
—— carbohydrate, 119, 120, 125
—— fat, 24, 119, 125, 128, 363
—— —— acid dermatitis and, 373
—— —— acute, 128
—— —— and dermatitis, 373
—— —— chronic, 129
—— —— clinical picture in, 129
—— —— treatment of, 129
—— in older children, 128
—— intestinal, and prolapse of rectum, 186
Dyspepsia, nervous, 129, 138
—— starch, 24, 130
—— —— abdomen in, 13, 130, 131
—— —— and constipation, 138
—— —— stools in, 131
—— —— treatment of, 131
Dyspnœa due to pulmonary tuberculosis, 421
Dystrophies, muscular, 315
Dystrophy, pseudo-hypertrophic, muscular, 316

E

Ear discharge, 149
—— —— chronic, in diphtheria, 404
—— diseases of, 146
—— —— (*see also* Mastoiditis ; Otitis media)
—— eczema of, causing otorrhœa, 149
—— epidemic infections and, 146
—— foreign bodies in, 146, 149
Earache, 146
Ears, examination of, 9
—— wax in, removal of, 10
Ectopia vesicæ, 255
Eczema, 384
—— anaphylaxis theory of, 384
—— and asthma, 219, 221
—— and mucous colitis, 178
—— and skin eruptions during dentition, 31
—— causing otorrhœa, 149
—— clinical picture in, 385
—— constitutional diathesis, theory of, 385
—— developing from otitis media, 148
—— dry type of, 386
—— etiology of, 384
—— irritation of, 386
—— local inflammation theory of, 385
—— prognosis of, 388
—— scaly type of, 386
—— treatment of, 386
—— weeping type of, 386
Edebohl's operation, 279
Egg-yolk, 79
"Eledon," 87, 123
Emphysema following whooping-cough, 400
Empyema, 210, 435

Empyema, clinical picture in, 211
—— complicating broncho-pneumonia, 202
—— —— lobar pneumonia, 209, 210
—— complications of, 213
—— course of, 212
—— etiology of, 210
—— in the new-born, 37
—— leading to septic meningitis, 298
—— "necessitas," 212
—— pathology of, 211
—— pneumonia and, 210
—— prognosis of, 212
—— symptoms of, 211
—— temperature in, 211
—— treatment of, 213
—— types of, 211
Encephalitis as cause of convulsions, 342
—— epidemic, 283
—— —— age- and sex-incidence of, 283
—— —— cerebro-spinal fluid in, 285, 295
—— —— clinical picture in, 284
—— —— course of, 286
—— —— differential diagnosis of, 286, 291, 295
—— —— etiology and pathology of, 283
—— —— incubation period of, 284
—— —— mental changes in, 286
—— —— moral incorrigibility in, 286
—— —— ophthalmoplegia in, 285
—— —— photophobia in, 295
—— —— prognosis of, 286, 287, 295
—— —— resemblance of to chorea, 445
—— —— seasonal incidence of, 283
—— —— sequelæ of, 286
—— —— simulated by appendicitis and peritonitis, 285
—— —— —— by tuberculous meningitis, 285
—— —— sleep in, 28
—— —— squint in, 9
Encephalitis epidemic, symptoms of, 284, 295
—— —— treatment of, during acute stage, 287
—— —— —— during chronic stages, 288
—— in typhoid fever, 407
—— lethargica (*see* Encephalitis, epidemic)
—— measles, 395
—— post-measles, 288, 395
—— post-vaccinal, 288, 415
—— rheumatic (*see* Chorea)
Endocrine insufficiency, 26
Enema, olive oil, 127
—— rash, 384
Eno's Fruit Salts, 129, 134, 139, 152, 396
Enteric fever (*see* Typhoid fever)
—— intussusception, 180
Enteritis, epidemic (*see* Diarrhœa, infective)
Entero-colitis, acute, 178
—— —— differentiated from intussusception, 181
Enuresis, acidosis and, 263
—— and boils, 379
—— congenital abnormalities and, 262
—— contributing factors in, 263
—— diabetes and, 262
—— frequency of micturition and, 263
—— functional, 262
—— prognosis of, 263
—— psychological aspect of, 265
—— symptoms and diagnosis of, 263
—— treatment of, 263
Ephedrine in asthma, 222, 223
Epidemic cerebro-spinal meningitis (*see* Meningitis, cerebro-spinal)
—— encephalitis (*see* Encephalitis, epidemic)
—— enteritis (*see* Diarrhœa, infective)
—— infections, predilection of, for the ear, 146
—— parotitis (*see* Mumps)
Epilepsy, 322, 338
—— age-incidence of, 339
—— clinical picture in, 339
—— diagnosis of, 340

Epilepsy, diagnosis of, differential, 342
—— etiology and pathology of, 339
—— fits in, 339, 340
—— idiopathic, 339
—— nocturnal, as cause of night terrors, 358
—— of mother, breast-feeding and, 71
—— prognosis of, 342
—— treatment of an attack in, 343
—— —— dietetic, 343
—— —— general management of child in, 345
—— —— medicinal, 344
Epiphysitis, syphilitic, 480
Epispadias, 255
Epistaxis due to whooping-cough, 400
—— in purpura, 248
—— in typhoid fever, 406
—— tendency to, in congenital pulmonary stenosis, 230
Epithelial casts in parenchymatous nephritis, 275
Epstein's diet, 279
Erb's paralysis, 48
Ergosterol preparations, 104, 106
Erysipelas, 278, 371
Erythema nodosum, 381
—— —— treatment of, 382
—— pernio, 374
—— rheumatic, 448, 451
Erythemata, toxic, 381
Erythrœdema polyneuritis (*see* Pink disease)
Eustachian catheter, 149, 258
Evaporated milk, 65
Everyday condensed milk, composition of, 65
Examination of normal child, 7
Excoriation of buttocks (*see* Dermatitis)
Exercise, need of, 29
Exercises, in treatment of spinal curvature, 366
Exfoliative dermatitis, 37
Exhaustion, nervous (*see* Nervous exhaustion)
Exophthalmic goitre, 23, 146
Exophthalmos, 146
—— accompanying habit spasm, 354
Expectorant mixture, 197
Expression of breast-milk, 74, 75
Exudative diathesis, 178
—— —— and blepharitis, 145
—— —— and eczema, 385
Eye accommodation, loss of, in post-diphtheritic paralysis, 312
—— diseases of, 144
Eyelids, diseases of (*see* Blepharitis; Conjunctivitis; Ophthalmia neonatorum; Styes)
Eyes, abnormalities of, 8
—— cataract of, 9
—— colour of, 32
—— examination of, 8
—— in mongolism, 9
—— lesions of, due to syphilis, 482
—— shape of, 8
—— (*see also* Ophthalmia neonatorum; Squint)

F

Face, in rickets, 10, 101
—— of normal child, 10
—— thinness of, causes of, 10
Fainting fits due to excessive thyroid administration, 337
Fairbank splint, 48
Farina, 142
Fat child, 331
—— —— (*see also* Obesity)
—— content of milk, breast-, 62, 70
—— —— —— condensed, 65
—— —— —— cow's, 62, 70
—— —— —— dried, full-cream, 66
—— —— —— —— humanized, 67
—— —— —— —— low-fat, 66
—— —— of patent starchy foods, 67
—— dyspepsia (*see* Dyspepsia, fat)
Fat-soluble A vitamin, 105
—— D vitamin, 106
Fatigued stance, 363
Fats, excess of, causing dermatitis, 373
Faulty posture, 363
Feeding, artificial, 80
—— —— buttermilk in, 87
—— —— calculation of food requirements in, 84

Feeding, artificial, carbohydrate in, 82
—— —— choice of bottle for, 88
—— —— —— of food for, 87
—— —— indicated in breast-abscess, 74
—— —— lactic acid milk in, 86
—— —— —— —— —— (*see also* Lactic acid milk)
—— —— methods of, 83
—— —— of premature infant, 57
—— —— starch in, 83
—— —— sugar in, 82, 85
—— —— temperature of feed in, 88
—— —— water in, 83
—— —— with condensed milk mixture, 57, 64
—— —— with cow's milk, 80, 81, 83
—— —— —— —— and added sugar, 85
—— —— —— —— diluted, and added carbohydrate, 84
—— —— with full-cream dried milk, 85
—— —— with humanized cow's milk mixtures, 83
—— —— —— dried milk, 85
—— bottle (*see* Bottle)
—— breast-, 69
—— —— advantages of, to child, 71
—— —— amount of milk required by child in, 73, 76
—— —— contra-indications to, 71
—— —— diet of mother and, 73
—— —— lack of co-ordination in, 74
—— —— length of time necessary for, 73
—— —— menstruation and, 71
—— —— normal, 71
—— —— of premature infant, 56
—— —— overfeeding in, 78
—— —— —— treatment of, 79
—— —— prevented by defect in child, 74
—— —— —— —— in mother, 74
Feeding, breast-, time and frequency of, 72
—— —— underfeeding in, 75
—— —— —— diagnosis of, 76
—— —— —— —— symptoms of, 75
—— —— with breast-abscess, 74
—— —— (*see also* Milk, breast-)
—— complementary, 71, 77
—— —— in breast-abscess, 74
—— —— rules for, 77
—— in congenital pyloric stenosis, 166
—— in dietetic diarrhœa, 125
—— mixed, commencement of, 79
—— —— —— of spoon-feeding in, 89
—— —— for children 9 months to 1 year old, 91
—— —— for infants 6 to 9 months old, 89, 90
—— —— night-feed in, 91
—— —— régime of, 89
—— —— starches in, 89
—— —— transition diet in, 91
—— —— when to commence, 89
—— post-operative, in pyloric stenosis, 167
—— supplementary, 77
—— test, 77
—— time and frequency of, 72
—— tube, 45, 59, 60, 287, 313
—— (*see also* Diet)
Fehling's test in examination of urine, 252
Fermentative diarrhœa, 120, 125
Fever, dehydration, 127
—— diet, 415
—— during dentition, 30
—— ear-drums in, 10
Fibroid lung, chronic, with bronchiectasis, complicating lobar pneumonia, 210
—— —— with bronchiectasis, 212
—— —— (*see also* Bronchiectasis)
Fibrolysin, 319
Fingers, clubbing of, in bronchiectasis, 215
—— —— in congenital heart disease, 229
—— tuberculosis of, 437
Finsen light in lupus vulgaris, 380
Fistula, fæcal, 428, 429

Fits (*see* Convulsions)
Flat-foot, 362
Flexner dysentery, 116, 118
Fomites, 390
Fontanelle, bulging of, 42, 44, 294
—— closure of, in hydrocephalus, 8
—— —— in microcephaly, 8
—— —— in osteogenesis imperfecta, 8
—— —— in premature infants, 8
—— —— in rickets, 99
—— depressed, in infective diarrhœa, 118
Food, solid, introduction of, 93
Food-tables for diabetes, 466, 470
Foods, patent starchy, 66
Force, 94, 134
Fracture, depressed, of skull, 45, 46
—— of ribs, 12
Fractures, in osteogenesis imperfecta, 368
Fragilitas ossium, 367
—— —— treatment of, 368
"Fragility test" in acholuric family jaundice, 234
Frequency of micturition (*see* Micturition, frequency of)
Fresh air, importance of, in pneumonia, 204
—— —— insufficiency of, a cause of disturbed sleep, 357
—— —— necessity for, in bronchitis, 197
—— —— need of for good health, 28
Friedreich's ataxia, knee-jerks in, 14
Fröhlich's syndrome, 19, 286
Fruit juice as source of vitamin C, 106, 109
—— —— in cœliac disease, 115
—— —— in infant feeding, 64, 90, 91, 92, 106, 109, 115, 272
—— —— in nephritis, 272
Full-cream condensed milks (*see* Milk, condensed, full-cream)
—— dried milk (*see* Milk, dried, full-cream)
Functional nervous diseases, 352
—— squint, 144
Furunculosis, 379
—— hyperglycæmia and, 379
—— treatment of, 379
Fusiform bacillus, 156, 158

G

Gait, limping, causes of, 13
—— rolling and staggering, causes of, 13
—— waddling, causes of, 13
Galactagogues, 75
Gangrene of lung, accompanying empyema, 214
Gargling, 152
Gastric attacks, 25, 133
—— influenza, 182, 411
—— lavage in pyloric stenosis, 166, 167, 168
Gastritis as cause of vomiting, 358
—— associated with pyloric stenosis, 163
—— chronic, due to dental caries, 160
Gastro-enterostomy in duodenal stenosis, 171
—— in duodenal ulcer, 172
Genito-urinary system, diseases of, 251
—— tuberculosis, 420
Geographical tongue, 159
German measles (*see* Rubella)
Gigantism, 27
Glands, cervical, enlargement of, due to dental caries, 160
—— —— —— due to tuberculosis (*see* Tuberculous cervical adenitis)
—— —— —— in infective mononucleosis, 154
—— —— —— in lymphadenoma, 243
—— —— —— in spleno-medullary leukæmia, 241
—— palpation of, 11
Glandular enlargement in acute lymphatic leukæmia, 240
—— —— in lymphadenoma, 243

Glandular enlargement in splenomedullary leukæmia, 241
—— —— significance of, 11
—— fever, 154
—— tuberculosis, 418, 421, 426, 433
Glaxo feeding-bottle, 88
—— full-cream dried milk, 66, 85, 88
—— half-cream dried milk, 66, 122
—— Malted Food, 68, 90
—— New, 67, 85, 88
Glax-ovo, composition of, 68
Glucose, in treatment of diabetes, 471
—— —— of epilepsy, 346
—— —— of fat dyspepsia, 130
—— —— of nervous exhaustion, 25
—— in urine in diabetes, 463
—— with saline in treatment of diarrhœa, 123, 127
Glycerin broth in tuberculin test, 438
—— instillation of, in otitis media, 148
Glycosuria, control of, with insulin, 466
—— renal, and diabetes, 464
Goat's milk, 62
Goitre, benign, 338
—— exophthalmic, 23, 146
Gonorrhœal ophthalmia, 39
—— vulvo-vaginitis, 269
Graded milks, 62
Grand mal, 339
Graves's disease, 23, 146
Green Butterfly condensed milk, composition of, 65
Greenstick fracture of ribs in osteogenesis imperfecta, 12
"Gripe-water" as cause of bromide rash, 383
Groats, 69, 79, 89, 90, 91, 92, 94, 134, 205
Groult's Cream of Rice, 68, 89, 90, 91, 92, 93, 114
"Growing pains," 449, 458
Growth in height (*see* Height)
—— in weight (*see* Weight)
—— normal and abnormal, 7
—— vitamin A and, 105
—— —— B and, 105
Gummata, miliary, 482
Gums, examination of, 10
—— in scurvy, 108

H

Habit spasm, clinical picture in, 353
—— —— etiology of, 354
—— —— treatment of, 354
Habits, good, training of children in, 15
—— of mother, and flow of breast-milk, 75
Hæmatemesis in Banti's disease, 233
Hæmatoma of scalp, 8, 45
—— of sternomastoid, 11, 46
Hæmaturia, characteristic of nephritis, 271
—— due to blood diseases, 255
—— due to calculus, 254
—— due to renal tuberculosis, 254
—— due to scurvy, 254
—— X ray examination in, 254
Hæmic bruit, 227
—— murmur in chlorosis, 239
Hæmoglobinuria, paroxysmal, 255
—— —— clinical picture in, 255
—— —— congenital syphilis and, 255
—— —— due to syphilis, 483
—— —— treatment of, 255
Hæmophilia, age and, 249
—— and purpura hæmorrhagica, differences between, 249
—— clinical picture in, 249
—— coagulation time in, 250
—— diagnosis of, 249
—— etiology of, 249
—— pathology of, 249
—— prognosis of, 250
—— sex and, 249
—— treatment of, 250
Hæmoptysis due to tuberculosis, 421
—— in bronchiectasis, 217
Hæmorrhage, cerebral, at birth, 41
—— from bowel, in typhoid fever, 407, 408
—— in scurvy, 107
—— into ventricle, causing hydrocephalus, 327 ?

Hæmorrhage, petechial, in acute lymphatic leukæmia, 240
—— —— in spleno-medullary leukæmia, 241
—— sub-periosteal, in scurvy, 108
—— suprarenal, in post-diphtheritic paralysis, 313
Hæmorrhagic disease of newborn, 49
—— nephritis (*see* Nephritis, hæmorrhagic)
Hæmorrhoids, 138
Hair, colour of, 32
—— in normal infants, 8
—— in various diseases, 8
Hare-lip, 50
—— and cleft palate preventing breast-feeding, 74
Harrison's sulcus, 12, 99
Hay fever and asthma, 219
Head, banging of, in dentition, 31
—— circumference of, 7
—— examination of, 7
—— protection of, 17
—— retraction in meningitis, poliomyelitis and epidemic encephalitis, 11, 295
—— —— significance of, 11
—— shape of, 8
Headache in typhoid fever, 406
Hearing, tests of, 9
Heart, auscultation of, 12
—— average rate of beat of, 225
—— congenital malformation of, 226, 227
—— dilatation of, 226
—— —— causes of, 226, 227
—— —— following pneumonia, 226
—— —— —— typhoid fever, 226
—— —— slight, 227
—— —— X-ray examination in, 226
—— disease, 225
—— —— and tendency to bronchitis, 195
—— —— congenital, 50, 229
—— —— —— accompanying mongolism, 231, 323, 325
—— —— —— cardiac hypertrophy in, 227
Heart disease, congenital, clubbing of fingers in, 231
—— —— —— failure to gain in weight in, 232
—— —— —— in premature infants, 60
—— —— —— murmurs in, 230
—— —— —— paroxysmal tachycardia in, 226
—— —— —— patent ductus arteriosus, 230
—— —— —— —— foramen ovale, 230
—— —— —— —— inter-ventricular septum, 230
—— —— —— preventing breast-feeding, 74
—— —— —— pulmonary stenosis, 229
—— —— —— tendency to constipation in, 232
—— —— —— treatment of, 231
—— —— —— types of, 229
—— —— lips in, 10
—— —— mortality of, 33, 34
—— —— prognosis of, 231
—— —— valvular, 227
—— —— (*see also* Carditis; Pericarditis; Pericardium, adherent)
—— hypertrophy of, 227, 280, 452
—— increased action of (*see* Tachycardia)
—— murmurs, significance of, 227, 228, 452
—— percussion of, 12
Heart-block in post-diphtheritic paralysis, 315
"Heat spots," 383
"Heating" foods, 382
Heels, wedging of, in flat-foot and knock-knee, 363
Height, factors retarding growth in, 26
—— growth in, 25
Heine-Medin disease (*see* Poliomyelitis, acute anterior)
Hemihypertrophy, head in, 8
Hemiplegia due to acute anterior poliomyelitis, 304

Hemiplegia, due to polio-encephalitis, 307
Henoch's purpura (*see* Purpura, Henoch's)
Hepatitis, acute, 36
—— —— (*see also* Jaundice, catarrhal)
Hereditary diseases, 32
Heredity in asthma, 219
—— in hæmophilia, 249
—— in rheumatism, 442
—— in tendency to acidosis, 132
—— influence of, 32
Hernia, 50, 188
—— accompanying hydrocele, 269
—— inguinal, 188
—— —— clinical picture in, 189
—— —— hydrocele resembling, 189
—— —— irreducible, 189
—— —— prognosis of, 189
—— —— sex-incidence of, 188
—— —— strangulated, 189
—— —— treatment of, 189
—— umbilical, 190
—— —— in cretinism, 190
—— —— in mongolism, 190, 324
—— —— treatment of, 190
Herniæ, combined inguinal and umbilical, 190
Herpes, accompanying lobar pneumonia, 208
—— febrilis, 381
—— of fifth nerve, 380
—— on lips, 10
—— recurrens, 381
—— zoster, 380
—— —— chicken-pox and, 380
—— —— symptoms of, 380
—— —— treatment of, 381
Herter's infantilism, 110
Hiccough, epidemic, in encephalitis lethargica, 286
Higginson's syringe, 147, 149
Hip, dislocation of, congenital, 13, 50, 362
—— —— —— diagnosis of, 362
—— —— —— treatment of, 362
—— tuberculosis of, 437
Hives, 383
Hodgkin's disease (*see* Lymphadenoma)
Hooker's Malted Milk, 66, 180
Horlick's Malted Milk, 66, 133, 135, 180, 223
Hot weather, breast-milk requirements in, 77
Human milk (*see* Milk, breast-)
Humanized condensed milk mixture, 57, 59, 64
—— dried milk, 58, 66, 67, 85, 88
"Humming-top" murmur, 230
Hunger pains, 28, 357
—— stools, 75
Hutchinsonian teeth, 483, 484
Hutchison's renal sarcoma, causing protrusion of eyeball, 146
Hydrocele, 189, 269
Hydrocephalus, 327
—— acquired, 328
—— clinical picture in, 329
—— communicating form of, 328
—— congenital, 322, 327
—— diagnosis of, 331
—— due to syphilis, 481
—— etiology and pathology of, 327
—— external, 327
—— following postbasic meningitis, 322, 330
—— head in, 8, 330
—— internal, 327
—— —— following cerebro-spinal meningitis, 293
—— mentality in, 331
—— non-communicating form of, 327
—— obstructive type of, 322
—— treatment of, 332
—— Wassermann reaction in, 332
—— with obesity, 19
Hydronephrosis, congenital, 255
—— due to kinking of ureters, 257
—— intermittent, simulating appendicitis, 175
Hyperæsthesia, in tuberculous meningitis, 290
Hyperglycæmia and boils, 379
Hyperpyrexia, convulsions due to, 350
Hyperthyroidism accompanying habit spasm, 354
Hypochlorhydria, 86
Hypoglycæmia as cause of disturbed sleep, 358

Hypoglycæmia in diabetes, 464, 469, 470, 471
Hypospadias, 255
Hypotonia, 12, 133, 310, 324, 363

I

Icterus neonatorum, 37
—— —— differential diagnosis of, 38
—— —— etiology of, 37
Ideal condensed milk, composition of, 65
—— —— —— mixture, 57
Idiocy, 41, 322
—— amaurotic family, 48
Ileocæcal intussusception, 180
Ileocolic intussusception, 180
Immune bodies in colostrum, 69
Immunity, breast-milk and, 71
Immunization in diphtheria, 405
—— in scarlet fever, 393
—— results of, general, 389
Impetigo bullous, 39, 378
—— contagiosa, 378
Incubator, improvised, 54, 55
Indigestion (*see* Dyspepsia)
Infant mortality (*see* Mortality)
Infantile paralysis (*see* Poliomyelitis, acute anterior)
—— scurvy (*see* Scurvy, infantile)
Infantilism, 26
—— due to congenital heart disease, 231
—— due to interstitial nephritis, 280
—— Herter's, 110
—— intestinal, 110
Infections, as cause of convulsions, 348
—— causing acidosis, 132
—— nose and throat (*see* Nose and throat infections)
—— of urinary tract, 256
Infectious diseases, 389
—— —— contacts in, 389, 390
—— —— incidence of, in childhood, 389
—— —— mortality of, in childhood, 389
—— —— quarantine periods and rules to be observed in, 390
—— fevers, acute, mortality of, 389
Infectious fevers, diet in, 415
Infective diarrhœa (*see* Diarrhœa, infective)
—— mononucleosis, 154
Influenza, 410
—— blood-count in, 412
—— clinical picture in, 411
—— complications of, 413
—— diagnosis of, 412
—— etiology and pathology of, 411
—— gastric, 182, 411
—— mortality of, 33
—— nervous symptoms in, 412
—— possible effects of, on heart, 226
—— prognosis of, 412
—— respiratory system in, 411
—— temperature in, 411
—— treatment of, 412
—— —— prophylactic, 413
Influenzal meningitis (*see* Meningitis, influenzal)
Inguinal and mesenteric adenitis, 174
—— hernia (*see* Hernia, inguinal)
Injuries of the new-born, 36, 41
Institutional treatment not necessary for premature infants, 55
Insulin in diabetes (*see* Diabetes mellitus, insulin in)
Intercostal drainage in empyema, 213
Interstitial keratitis, 144, 482, 484
—— nephritis (*see* Nephritis, interstitial)
—— pneumonia (*see* Pneumonia, interstitial)
Intestinal indigestion, 186
—— infantilism, 110
—— parasites, 472
Intoxication in infective diarrhœa, 118
Intracutaneous tuberculin test (*see* Tuberculin test, intracutaneous)
Intranasal oxygen, in dietetic diarrhœa, 125
Intraventricular indicator, use of, 331
Intubation in croup, 192, 193
—— in laryngeal papilloma, 194
Intussusception, 180

Intussusception, chronic, 181
—— —— and tuberculous peritonitis, 430
—— —— simulating appendicitis, 175
—— clinical picture in, 181
—— differential diagnosis of, 181
—— etiology of, 180
—— forms of, 180
—— prognosis of, 181
—— treatment of, 181
Ipecacuanha, as cause of vomiting, 358
Iritis, accompanying herpes of 5th nerve, 380
Iron-and-copper mixture in chlorosis, 239
Iron deficiency in premature infants, 61
Irradiated ergosterol preparations, 104

J

Jaundice, acholuric family, 234
—— —— —— etiology and pathology of, 234
—— —— —— prognosis of, 236
—— —— —— symptoms and diagnosis of, 234
—— —— —— treatment of, 236
—— —— —— typical example of, 236
—— catarrhal, 182
—— —— clinical picture in, 182
—— —— complications of, 183
—— —— diet in, 183
—— —— etiology and pathology of, 182
—— —— liver in, 13
—— —— treatment of, 183
—— of new-born (*see* Icterus neonatorum)
—— septic, acute, due to umbilical sepsis, 36
Joints, diseases of, 362
—— rheumatism of (*see* Rheumatism, arthritis in)
—— tuberculosis of, 418

K

Kalzana, 375
Kaolin in treatment of dietetic diarrhœa, 124
Keratitis, interstitial, 144, 482, 484
Kernig's sign, in cerebro-spinal meningitis, 293
—— —— in tuberculous meningitis, 290
Ketogenic diet, in treatment of epilepsy, 343
Ketosis, 82
—— (*see also* Acidosis)
Kidney, congenital cystic, 26, 255
—— decapsulation of, 279
—— disease and tendency to bronchitis, 195
—— fibrotic, in parenchymatous nephritis, 278
—— polycystic, 279
—— tuberculosis of, 418
Kindergarten advantageous for diabetic child, 465
—— —— for nervous-exhaustion child, 25, 353
—— —— for subjects of cyclical vomiting, 134
Klebs-Loeffler bacillus, 403
Klim full-cream dried milk, composition of, 66
—— skimmed dried milk, composition of, 66
Knee, tuberculosis of, 437
Knee-jerks, absence of, 14
—— examination of, 14
—— "hung up," in chorea, 446
—— increased, 14
—— loss of, in post-diphtheritic paralysis, 312
Knees, swellings of, due to syphilis, 484, 485
Knock-knee, 13, 362
Koch's Old Tuberculin, 438
Koch-Weeks bacillus, 144
Koplik's spots, 395
Kyphosis, 12, 352, 364

L

Lacta dried milk, 66, 85
Lactalbumin, 70, 81
Lactation, establishment of, 72
—— maintenance of, 74
—— measures for improving, 75
—— menstruation and, 71
—— stimulation of, by suction, 75
—— (*see also* Feeding, breast-; Milk, breast-)
Lactglobulin, 70

Lactic acid milk in infant feeding, 86
—— —— —— in cœliac disease, 114
—— —— —— in dietetic diarrhœa, 122, 125
—— —— —— preparation of, 58, 86, 122
Lactogen dried milk, composition of, 66
Lactose, 70, 82, 85, 137
Laparotomy in duodenal stenosis, 171
Laryngeal diphtheria, differentiated from spasmodic croup, 192
—— papilloma, 194
—— —— differentiated from congenital laryngeal stridor, 193
—— stridor, congenital, 193
—— —— —— treatment of, 194
Laryngismus stridulus, 102
—— —— differentiated from congenital laryngeal stridor, 193
—— —— —— from spasmodic croup, 192
Laryngitis, 151, 191
—— catarrhal, 191
—— complicating chicken-pox, 402
—— diphtheritic, 191
—— syphilitic, 480
Lateral sinus infection, complicating mastoid disease, 149
—— —— thrombosis, accompanying empyema, 214
Legs, abnormally short, 13
—— bowed, 13
—— knock-kneed, 13, 362
—— shape of, 13
Length of infant (*see* Height)
Leukæmia, acute lymphatic, 240
—— —— —— age- and sex-incidence of, 240
—— —— —— etiology and pathology of, 241
—— —— —— prognosis of, 241
—— —— —— symptoms and diagnosis of, 240
Leukæmia, acute lymphatic, treatment of, 241
—— differentiated from lymphadenoma, 246
—— hæmaturia in, 255
—— myelogenous (*see* Leukæmia, spleno-medullary)
—— spleno-medullary, 241
—— —— age-incidence of, 241
—— —— blood-picture in, 242
—— —— etiology of, 242
—— —— pathology of, 242
—— —— prognosis of, 242
—— —— symptoms and diagnosis of, 241
—— —— treatment of, 242
—— —— von Jaksch's anæmia and, 238
Leukæmias, mixed, 242
Leukanæmia, lymphatic, 241
Libby's condensed milk, composition of, 65
—— —— —— mixture, 57
Lichen urticatus, 31, 375, 382
Limping, causes of, 13
Lipiodol in bronchiectasis, 216
Lipodystrophia progressiva, face in, 10
—— —— thinness in, 24
Lips, examination of, 10
Liston splint, 187
Little's disease (*see* Spastic paralysis)
Liver, administration of, in hæmophilia, 250
—— chill on, 182
—— cirrhosis of, 13, 38, 185
—— —— differentiated from tuberculous peritonitis, 430
—— —— due to syphilis, 482, 484
—— —— infective, 185
—— —— with ascites, 186
—— enlargement of, due to syphilis, 482
—— —— due to tuberculosis, 13
—— —— in glandular fever, 154
—— —— in lymphadenoma, 243
—— —— significance of, 13
—— fatty, in infective diarrhœa, 118
—— in cœliac disease, 13, 110, 113
—— normal size of, 13

Liver, palpation of, 13
—— ptosis of, 13
—— (*see also* Hepatitis)
Lockjaw, 38
Lumbar puncture, 319
—— —— amount of fluid to be withdrawn in, 320
—— —— in cerebro-spinal meningitis, 296
—— —— in epidemic encephalitis, 287
—— —— in influenzal meningitis, 300
—— —— in measles, 396
—— —— in miliary tuberculosis, 425
—— —— in septic meningitis, 299
—— —— in spastic paralysis, 44
—— —— in treatment of convulsions, 351
—— —— in tuberculous meningitis, 291, 292
—— —— preparation of patient for, 319
—— —— technique of, 320
Lung, atelectasis of, congenital, 52
—— —— —— as cause of retarded growth, 26
—— fibroid (*see* Fibroid lung; Bronchiectasis)
—— fibrosis of, following whooping-cough, 400
Lungs, examination of, 12
—— tuberculosis of (*see* Tuberculosis, pulmonary)
Lupus vulgaris, 380, 418
—— —— treatment of, 380
Lymphadenoma, 243
—— abdominal type of, 244, 430
—— age-incidence of, 243
—— diagnosis of, 243, 246
—— etiology and pathology of, 246
—— generalized type of, 244
—— prognosis of, 247
—— symptoms of, 243
—— temperature in, 245, 246
—— treatment of, 247
Lymphatic leukæmia, acute (*see* Leukæmia, acute lymphatic)
—— leukanæmia, 241
Lymphosarcoma, 246

M

Macerated fœtus due to syphilis, 479
Magnesia, milk of, 129, 134, 136, 137, 152, 158, 161
Malformation of nipples, preventing breast-feeding, 74
—— of vertebræ, 12, 364
Malformations, congenital, 49
—— —— etiology and pathology of, 50
—— —— treatment of, 50
—— —— of urinary tract, 255
Malnutrition due to dental caries, 159
Malted foods, value of, in maintaining lactation, 75
Mammals, various, milk of, 62
Mantoux test (*see* Tuberculin test, intracutaneous)
Marasmus, face in, 10
Marmite, 105
Marriott's method for preparing lactic acid milk, 86, 122
Martindale, W., 265
Massage in constipation in infants, 136
—— in flat-foot and knock-knee, 363
—— in starch dyspepsia, 131
—— of breasts, 75
Mastoiditis, acute, 148
—— —— clinical picture in, 148
—— —— complications of, 149
—— —— etiology of, 148
—— —— œdema in, 149
—— —— treatment of, 149
—— and otitis media, 147
—— differentiated from tuberculous meningitis, 291
—— fever diet in, 415
—— prevention of, 149
Masturbation, 268, 359
Mead's Dextrimaltose, 82, 85
Measles, 394
—— accompanied by nose and throat infections, 150
—— as cause of anorexia, 355
—— clinical picture in, 395
—— complications of, 146, 200, 203, 395
—— encephalitis, 395
—— German (*see* Rubella)

Measles, incubation period of, 394, 396
—— influence of on incidence of tuberculosis, 424
—— Koplik's spots in, 395
—— leading to bronchiectasis, 214
—— mortality of, 33, 34, 389
—— —— in children of school and pre-school age, 389
—— period of infectivity in, 390
—— quarantine period and rules to be observed in, 390
—— rash of, 395
—— return to school after exposure to infection of, 390
—— temperature in, 395
—— treatment of, 396
Meat juice, raw, 61
Meatal ulcer, 267
—— —— as cause of disturbed sleep, 358
Meatotomy, 268
Meatus, pin-point, 268
Meckel's diverticulum, adherent, as cause of volvulus, 182
—— —— —— simulating appendicitis, 175
Mediastinal-gland tuberculosis (*see* Tuberculosis, mediastinal-gland)
Medical Officers of Schools Association, leaflet of, 390
Melæna, 97
—— due to duodenal ulcer, 171
—— due to tuberculous ulceration of intestine, 429
—— neonatorum, 37, 49, 97
Mellin's Food, 68, 82, 137
Meningism, 209
Meningitis, cerebro-spinal, 292
—— —— accompanied by herpes febrilis, 381
—— —— age-incidence of, 48, 292
—— —— cerebro-spinal fluid in, 295
—— —— cistern puncture in, 296
—— —— clinical picture in, 293
—— —— diagnosis of, 294, 295
—— —— etiology and pathology of, 292
—— —— head retraction in, 294, 295
Meningitis, cerebro-spinal, in older children, 293
—— —— leading to internal hydrocephalus, 293
—— —— mortality of, 33
—— —— prognosis of, 294, 295
—— —— symptoms of, 294, 295
—— —— treatment of, 294
—— —— —— of fulminating type of, 294
—— —— —— of mild type of, 294
—— —— —— of postbasic type of, 296
—— —— types of, fulminating, 293
—— —— —— mild (Type 3), 293
—— —— —— postbasic, 294
—— complicating lobar pneumonia, 209
—— —— mastoid disease, 149
—— —— mumps, 397
—— differentiated from epidemic encephalitis, 286
—— in new-born, 37
—— influenzal, age and, 48
—— —— cerebro-spinal fluid in, 300
—— —— clinical picture in, 300
—— —— etiology and pathology of, 299
—— —— organism of, 299
—— —— treatment of, 300
—— meningococcal (*see* Meningitis, cerebro-spinal)
—— mumps, 397
—— pneumococcal (*see* Meningitis, septic)
—— postbasic, 294
—— —— leading to hydrocephalus, 322
—— septic, 297
—— —— cerebro-spinal fluid in, 295
—— —— clinical picture in, 298
—— —— conditions leading to, 298
—— —— diagnosis of, 299
—— —— etiology of, 297
—— —— lumbar puncture in, 299
—— —— middle-ear infection and, 297
—— —— organism of, 297
—— —— prognosis of, 295, 298

Meningitis, septic, resemblance of, to influenzal meningitis, 300
—— —— symptoms of, 295
—— —— treatment of, 299
—— simulated by otitis media, 147
—— —— in pyelitis, 258
—— squint in, 9
—— streptococcal (*see* Meningitis, septic)
—— suppurative, accompanying empyema, 214
—— syphilitic, 481
—— —— as cause of hydrocephalus, 322, 482
—— tuberculous, 288, 423, 425
—— —— age-incidence of, 288
—— —— cerebro-spinal fluid in, 291, 295
—— —— course and duration of, 291
—— —— diagnosis of, 290, 295
—— —— etiology and pathology of, 289
—— —— hyperæsthesia in, 290
—— —— mode of infection in, 289
—— —— post-mortem appearances in, 289
—— —— prevention of, 292
—— —— prognosis of, 291, 295
—— —— resemblance of to acute anterior poliomyelitis, 304
—— —— seasonal incidence of, 289
—— —— stages of, 290
—— —— symptoms of, 290, 295
—— —— treatment of, 292
Meningococcal meningitis (*see* Meningitis, cerebro-spinal)
—— septicæmia, 293
Menstruation and lactation, 71
Mental defect, acquired, 322, 333
—— —— congenital, 322, 323, 326
—— —— due to syphilis, 482
—— —— following epidemic encephalitis, 284, 286
—— —— forms of, 322
—— —— primary, 322
—— —— secondary, 322, 333
Mercury tube in treatment of cardiospasm, 162
Merycism (*see* Rumination)
Mesentery, dragging on, simulating appendicitis, 175
Metabolic estimations in cretinism, 337
Methyl violet solution, injection of, in hydrocephalus, 331
Microcephaly, 41, 322, 326
—— etiology of, 326
—— head in, 8
—— pathology of, 326
—— prognosis of, 326
—— treatment of, 326
Micturition, frequency of, 16, 263, 266
—— —— causes of, 266, 462
—— —— treatment of, 266
—— —— (*see also* Enuresis)
—— training of child in good habits of, 15, 266
Mid-diastolic murmur in rheumatic carditis, 228, 452
Migraine, 132, 219
Miliary tuberculosis (*see* Tuberculosis, miliary)
Milk as source of infection in infantile paralysis, 301
—— —— —— in tuberculosis, 417
—— —— of vitamin D, 106
—— ass's, 62
—— bottled, 63
—— breast-, amount of required, 76
—— —— and immunity, 71
—— —— casein in, 62, 70
—— —— compared with cow's milk, 70, 80
—— —— composition of, 70
—— —— —— variations in, 70
—— —— digestion of, 70
—— —— effect of drugs on, 73
—— —— expression of, 56, 74
—— —— failure of infant to thrive on, 80
—— —— fat content of, 62, 70
—— —— flow of, establishment of, 72
—— —— —— methods of increasing, 75
—— —— —— stimulation of, 72
—— —— lactalbumin in, 70
—— —— lactglobulin in, 70
—— —— lactose in, 70

Milk, breast-, protein of, 70
—— —— quality of, variations in, 78
—— —— quantity and quality of, variations in, 72
—— —— salts in, 70, 71
—— certified, 62
—— condensed, 63
—— —— and vitamin A, 105
—— —— curds of, 81
—— —— full-cream, sweetened, 63
—— —— —— —— composition of, 65
—— —— —— unsweetened, 64
—— —— —— —— composition of, 65
—— —— humanized, 57, 59, 64
—— —— in treatment of pyloric stenosis, 168
—— —— skimmed, sweetened, 64
—— —— —— —— composition of, 65
—— —— varieties of, 63
—— cow's, albumin in, 62, 70
—— —— boiled, advantages and disadvantages of, 87
—— —— boiling of, effect of, 63, 81, 87, 107
—— —— —— —— on vitamin A, 105
—— —— calculation of requirements of in infant-feeding, 84
—— —— carbohydrate of, 82
—— —— casein of, 62, 70, 81
—— —— compared with human, 70, 80
—— —— composition of, 70
—— —— curd of, 81
—— —— —— modification of, 63, 81
—— —— dilution of, effects of, 70, 81
—— —— effects of excessive protein in, 18
—— —— fat of, 70, 82
—— —— in winter deficient in vitamins, 102
—— —— lactose in, 70, 82
—— —— modification of for in fant feeding, 81
—— —— peptonization of, 81
—— —— protein of, 70, 82
Milk, cow's, salts in, 70, 71
—— —— sugar to be added to, 85
—— —— —— present in, 70, 82
—— —— unboiled, advantages and disadvantages of, 87
—— —— vitamins in, 63, 64, 87, 105, 106, 107
—— diet, as cause of anorexia, 356
—— dried, 64
—— —— advantages and disadvantages of, 64, 81, 88
—— —— and vitamin A, 105
—— —— full-cream, composition of various brands of, 66
—— —— —— requirements of, in infant-feeding, 85
—— —— humanized, 58, 66
—— —— —— advantages and disadvantages of, 88
—— —— —— composition of various brands of, 67
—— —— —— requirements of, in infant-feeding, 85
—— —— in treatment of cœliac disease, 114
—— ——skimmed, composition of various brands of, 66
—— —— with low fat content, composition of, 66
—— goat's, 62
—— Grade A, 63
—— human (*see* Milk, breast-)
—— lactic acid (*see* Lactic acid milk)
—— mare's, 62
—— mixed, advantages of, 417
—— of different mammals compared, 62
—— pasteurized, 81, 105, 107
—— peptonized, 58, 81, 168
—— quantity of, for children of school age, 95
—— rich, excess of, causing mucous colitis, 179
—— sugar, 70, 82
—— tuberculin-tested, 63
—— "windy," 76
Milkal dried milk, 66, 85

Mineral salts in cow's milk, 70, 71
—— —— in human milk, 70, 71
Mitral regurgitation in rheumatic carditis, 228, 452
—— stenosis in rheumatic carditis, 228, 452
—— —— præcordial thrill in, 12, 228, 452
Mixed feeding (*see* Feeding, mixed)
Modification of cow's milk for infant feeding, 81
Molars (*see* Teeth)
Mongolism, 322, 323
—— and congenital heart defects, 231, 323, 325
—— and tendency to umbilical hernia, 190, 324
—— clinical picture in, 323
—— compared with cretinism, 335
—— etiology and pathology of, 50, 323
—— eyes in, 9
—— hair in, 8
—— mouth in, 10
—— prognosis of, 325
—— squint in, 9
—— treatment of, 325
Mononucleosis, infective, 154
Moonraker condensed milk, composition of, 65
Morbilli (*see* Measles)
Morbus cordis, congenital (*see* Heart disease, congenital)
Morgan bacilli in stools, 118
Mortality in childhood in 1928, principal causes of, 33, 34
—— infantile (1861-1927), 35
Motor control, development of, 14
—— paralysis following epidemic encephalitis, 286
Mouth, examination of, 10
Mumps, 396
—— clinical picture in, 397
—— complications of, 397
—— incubation period of, 396
—— period of infectivity in, 390
—— quarantine period and rules to be observed in, 390
—— return to school after exposure to infection of, 390
—— symptoms of, 397
—— treatment of, 397
Muscular atrophy, progressive spinal (*see* Werdnig-Hoffmann paralysis)
—— dystrophies, 315
Mustard-coloured stools, 96
Myelogenous leukæmia (*see* Leukæmia, spleno-medullary)
Myoclonus in epidemic encephalitis, 286
Myopathies, 315
Myositis, 448
Myxœdema, 338

N

Nævi, 50
Napkin rashes, 372
Nasal diphtheria, 404
—— feeding, 60
—— obstruction as cause of disturbed sleep, 357
—— —— nose in, 9
—— —— preventing breast-feeding, 74
—— oxygen (*see* Oxygen, nasal)
—— voice in post-diphtheritic paralysis, 313
Navel, condition of, 13
Neave's Cereal Food, composition of, 68
—— Milfo dried milk, composition of, 66
Nebulæ, opaque, resulting from conjunctivitis, 145
Neck glands (*see* Glands, cervical)
—— palpation of, 11
—— stiffness of, 11, 450
Negativistic child subject to anorexia, 355
Nephritis, 261, 271
—— breast-feeding in, 71
—— causing hæmaturia, 254
—— complicating nose and throat infections, 151
—— glomerular, 273
—— hæmorrhagic, acute, age- and sex-incidence of 272
—— —— —— etiology and pathology of, 271
—— —— —— hæmaturia in, 271
—— —— —— prognosis of, 272
—— —— —— symptoms and diagnosis of, 271
—— —— —— treatment of, 272

Nephritis, hæmorrhagic, acute, urine in, 271
—— —— complicating scarlet fever, 392
—— interstitial, 261, 278, 279
—— —— and enuresis, 262
—— —— as cause of wasting, 24, 26
—— —— blood-urea in, 253, 280
—— —— diagnosis of, 280
—— —— —— Wassermann reaction in, 282
—— —— etiology and pathology of, 279
—— —— prognosis of, 280
—— —— renal dwarfism and, 279, 280, 281
—— —— rickets due to, 280
—— —— stature in, 280
—— —— symptoms and diagnosis of, 280
—— —— syphilis and, 282
—— —— treatment of, 282
—— —— urine in, 280
—— mixed parenchymatous and glomerular, 273
—— mortality of, 33, 34
—— parenchymatous, 273, 274
—— —— accompanied by pyelitis, 277
—— —— acute, 274, 275, 279
—— —— complicated by anuria, 276
—— —— etiology and pathology of, 274
—— —— following scarlet fever, 392
—— —— history of sore throat in, 274
—— —— prognosis of, 276
—— —— stages of, 275
—— —— subacute and chronic, 276, 278
—— —— symptoms and diagnosis of, 274
—— —— treatment of, 276
—— —— urine in, 274
—— post-scarlatinal, 273
—— septic tonsils and, 151
—— syphilitic, 282, 483
—— —— as cause of convulsions, 351
—— tubular, 261, 273
Nephrosis (*see* Nephritis, parenchymatous)
Nervous child and frequency of micturition, 267
—— diseases, 283
—— —— functional, 352
—— —— mortality of, 33
—— dyspepsia, 129
—— —— and tendency to constipation, 138
—— exhaustion child, 24, 129, 138, 352
—— —— —— and tendency to albuminuria, 254
—— —— —— —— to constipation, 138
—— —— —— appearance of, 352
—— —— —— management of, 353
—— —— posture in, 363
—— infant, breast-milk requirements of, 77
—— rickets, 102, 350
—— system, rheumatism of (*see* Chorea)
—— tachycardia, 225
—— vomiting, 358
Nervousness, due to dental caries, 160
Nestlé's condensed milk, composition of, 65
—— Milk Food, composition of, 68
Nettle rash, 383
Neuralgia as cause of disturbed sleep, 357
—— during dentition, 28, 31, 357
Neuritis, optic and epidemic encephalitis, 287
—— peripheral, 309, 312
—— —— differentiated from epidemic encephalitis, 287
—— —— in typhoid fever, 407
—— —— sensory, 309
—— post-diphtheritic, differentiated from epidemic encephalitis, 287
New growths, (*see* Tumours)
—— Zealand, infant mortality in, 35
New-born, diseases of, 36

New-born, hæmorrhagic disease of, 49
—— injuries of, 36
Night sweats, significance of, 421
—— terrors, causes of, 27, 357
Nipples, cracked or depressed, 74
—— preparation of, 69
" Nits," 376
Nocturnal wakefulness, 28, 286
Nodules, rheumatic, 448, 450
" Noma," 157
Normal child, examination of, 7
—— —— feeding of, 62
—— saline, preparation of, 126
—— stools, composition of, 96
Nose and throat infections, 149
—— —— —— and asthma, 151, 219, 221
—— —— —— clinical picture and symptoms of, 150
—— —— —— complications of, 151
—— —— —— etiology of, 150
—— —— —— followed by nephritis, 271
—— —— —— organisms concerned in, 150
—— —— —— pathology of, 150
—— —— —— septic tonsils and adenoids in, 152
—— —— —— treatment of, 152
—— —— —— Vincent's angina, 150
—— diseases of, 144
—— shape of, 9
—— saddle-shaped, due to syphilis, 484, 485
Nose-finger test, 14
Nursing mother, diet of, 73
Nutrition, diseases of, 96

O

Obesity, causes of, 19
—— diet in, 20, 21
—— distribution of fat in, 20
—— flat-foot in, 17, 363
—— prognosis of, 22
—— treatment of, 20
Œsophageal obstruction as cause of vomiting, 358
—— stricture or stenosis, 162
Œsophagectasia(*see* Cardiospasm)
Oïdium albicans, 155
Olive-oil enema, in diarrhœa, 127
—— unsuitable in constipation, 137
" Only " children, liable to nervous exhaustion, 353
Open-air schools for subjects of rheumatic carditis, 455
" Opening Doors " (Thomson), 325
Ophthalmia neonatorum, 39, 144
—— —— clinical picture in, 39
—— —— etiology of, 39
—— —— treatment of, 39
Ophthalmoplegia, 285
—— (*see also* Squint)
Orbit, inflammation of, 146
Orchitis, complicating mumps, 397
Organic squint, 144
Organotherapy, in pseudo-hypertrophic muscular paralysis, 319
Orthostatic albuminuria (*see* Albuminuria, orthostatic)
Osteitis, syphilitic, 481
Ostelin, 104, 106, 357
Osteogenesis imperfecta, 368
—— —— fontanelle in, 8
—— —— fractures in, 12, 368
Osteomyelitis, 369
—— age-incidence of, 369
—— and scurvy, 108
—— causative organism in, 369
—— clinical picture in, 370
—— differential diagnosis of, 371
—— mortality of, 33
—— of ribs in empyema, 213
—— of turbinate bones, 146
—— treatment of, 371
Osteoporosis due to syphilis, 480
—— in cœliac disease, 110
—— in osteogenesis imperfecta, 368
Ostomalt, 106
Otitis media, 146, 151
—— —— clinical picture in, 146
—— —— complicating diphtheria, 146
—— —— —— influenza, 146,411, 413
—— —— —— measles, 146, 395
—— —— —— nose and throat infections, 151
—— —— —— scarlet fever, 146, 392
—— —— earache in, 146, 147
—— —— etiology of, 146

Otitis media, fever diet in, 415
—— —— giving rise to eczema, 148
—— —— leading to septic meningitis, 297
—— —— organisms concerned in, 147
—— —— prevention of, 149
—— —— prognosis of, 147
—— —— simulated by meningitis, 147
—— —— treatment of, 147
Otorrhœa, 149
Ovaltine, 68, 73
Ovarian tumour, 27
Overfeeding, 119
—— as cause of diarrhœa, 116, 119
—— as cause of disturbed sleep, 357
—— as cause of urticaria, 382
—— on breast, symptoms of, 76
—— —— treatment of, 77
—— on fats as cause of cœliac disease, 110
Over-active child, 18, 23, 354
Over-excitement and acidosis, 132
—— and asthma, 219, 220, 222
—— and enuresis, 263
Over-exertion and acidosis, 132
—— and asthma, 219, 220, 222
—— and enuresis, 263
Overgrowth, 27
Over-weight child and constipation, 135
—— —— and eczema, 386
—— —— (*see also* Obesity)
Oxygen, administration of, in "blue turns" in premature infants, 60
—— —— in miliary tuberculosis, 425
—— nasal, in dietetic diarrhœa, 125
—— —— in pneumonia, 204, 205
Oxyuris vermicularis, 472, 475

P

Pain, abdominal (*see* Abdominal pain)
—— on moving limbs, significance of, 13
Pains, hunger, 28
Palatal paralysis, 312, 315, 404
Palate, soft, syphilitic perforation of, 480, 481
Pallor, 10
Palpitation due to excessive administration of thyroid, 337
Pancreatitis, acute, complicating mumps, 397
Papilloma, laryngeal (*see* Laryngeal papilloma)
Paraldehyde, rectal injection of, for anæsthesia, 153
Paralysis, Erb's, 48
—— facial, 315
—— —— treatment of, 315
—— infantile (*see* Poliomyelitis, acute anterior)
—— palatal, 312, 315, 404
—— post-diphtheritic, 312, 404
—— —— clinical picture in, 312
—— —— complications of, 313
—— —— duration of, 313
—— —— etiology of, 312
—— —— gait in, 14
—— —— prognosis of, 313
—— —— treatment of, 314
—— pseudo-hypertrophic muscular, 33, 316
—— —— —— clinical picture in, 316
—— —— —— treatment of, 318
—— spastic (*see* Spastic paralysis)
—— Werdnig - Hoffmann (*see* Werdnig-Hoffmann paralysis)
Parasites, intestinal, 472
Parasitic eruptions, 375
Parathyroid in treatment of chilblains, 375
Paratyphoid fever, 410
—— —— differentiated from miliary tuberculosis, 425
—— —— temperature in, 409
—— organisms in stools, 118
Parenchymatous nephritis (*see* Nephritis, parenchymatous)
Parenteral infections (*see* Diarrhœa, symptomatic)
Parkinsonism, following epidemic encephalitis, 286
Parotitis, epidemic (*see* Mumps)
Paroxysmal hæmoglobinuria (*see* Hæmoglobinuria, paroxysmal)

Paroxysmal tachycardia, 226
Parrish's Chemical Food, 131, 239, 282, 357
Parrot's pseudo-paralysis, 480
Pasteurized milk, 81, 105, 107
Patent foods, starchy, composition of, 67
Pediculosis capitis, 376
—— —— associated with impetigo contagiosa, 378
Pel-Ebstein temperature in lymphadenoma, 246
Pellagra, 105
Pemphigus neonatorum, 39, 378
—— —— etiology and pathology of, 39
—— —— treatment of, 40
Peptonized milk, 58, 81, 168
Pericarditis in new-born, 37
—— leading to septic meningitis, 298
—— pneumococcal, 228
—— —— complicating lobar pneumonia, 209, 210
—— rheumatic, 228, 444, 453
—— suppurative, 228
Pericardium, adherent, 227, 228, 454
Periostitis, syphilitic, 484
Peripheral neuritis, 309, 312
Peritonitis in new-born, 37
—— complicating lobar pneumonia, 209
—— gonorrhœal, 270
—— leading to septic meningitis, 298
—— pneumococcal, 176, 270
—— —— complicating vulvo-vaginitis, 176, 270
—— —— diarrhœa in, 177
—— —— differentiated from appendicitis, 174
—— —— —— from tuberculous peritonitis, 430
—— —— operation in, 177
—— —— primary, 176
—— —— residual abscess in, 177
—— —— secondary, 178
—— —— septicæmia in, 177
—— septic, due to perforation of tuberculous ulcer, 428
—— simulated in pyelitis, 258
Peritonitis, simulated in epidemic encephalitis, 285
—— tuberculous (*see* Tuberculous peritonitis)
Peritonsillar abscess, 151
Permanent teeth, 32
Pertussis (*see* Whooping-cough)
Pet (condensed) milk, composition of, 65
Petit mal, 340
—— —— resemblance of, to paroxysmal tachycardia, 226
—— —— salaam type of, 340
Petrolagar, 136, 139
Pfeiffer's bacillus, 299, 411
Phenol-sulphone-phthalein as intraventricular indicator, 331
Phimosis, 13, 268
Phlyctenular conjunctivitis (*see* Conjunctivitis, phlyctenular)
Photophobia, due to phlyctenular conjunctivitis, 145
—— due to syphilis, 482
—— in meningitis, poliomyelitis and epidemic encephalitis, 295
—— in pink disease, 310
Pigeon chest, 12
Pineal gland, tumour of, 28
Pink disease, 309
—— —— age-incidence of, 309
—— —— and teething, 31
—— —— clinical picture in, 309
—— —— diet in, 312
—— —— etiology of, 311
—— —— mode of onset of, 309
—— —— pathology of, 312
—— —— prognosis of, 312
—— —— rash of, 310
—— —— regional incidence of, 311
—— —— symptoms of, 309
—— —— tachycardia in, 225, 310
—— —— treatment of, 312
—— eye, 394
" Pin-point " meatus, 13, 268
Pirquet test, 419
Pituitary gland in treatment of obesity, 22
—— tumour, 19
Pityriasis rosea, 388
Pleurisy, 435

Pleurisy, diaphragmatic, differentiated from appendicitis, 173
—— non-purulent, 435
—— purulent, 210, 435
—— —— (*see also* Empyema)
—— tuberculous, 435
—— —— pathology of, 435
—— —— primary focus in, 435
—— —— prognosis of, 435
—— —— symptoms and clinical picture of, 435
—— —— treatment of, 436
—— with effusion, 435
—— with empyema, complicating lobar pneumonia, 209
Pneumococcal meningitis (*see* Meningitis, septic)
—— peritonitis (*see* Peritonitis, pneumococcal)
Pneumonia, 198
—— age-incidence of, 198
—— albuminuria in, 253
—— breast-feeding in, 71
—— broncho-, 199
—— —— and lobar, differences between, 199
—— —— clinical picture in, 201
—— —— clothing in, 204
—— —— complicating whooping-cough, 400
—— —— complications of, 202
—— —— course and duration of, 203
—— —— etiology of, 199
—— —— leading to bronchiectasis, 214
—— —— pathology of, 200
—— —— physical signs of, 201
—— —— primary, 200
—— —— prognosis in, 202
—— —— resulting from diarrhœa, 118, 200
—— —— septic, terminating cancrum oris, 159
—— —— symptoms of, 201
—— —— temperature in, 201,203
—— —— —— falling by lysis in, 203
—— —— treatment of, 203
—— complicated by pneumococcal pericarditis, 228
—— —— by pneumococcal peritonitis, 178
Pneumonia, complicating measles, 395
—— congenital heart disease in, 203
—— examination of chest in, 201
—— fever diet in, 415
—— following parenchymatous nephritis, 278
—— —— surgical treatment of laryngeal papilloma, 194
—— hare-lip or cleft palate in, 203
—— herpes febrilis in, 381
—— in new-born, 37
—— interstitial, 211 212, 213, 214
—— —— (*see also* Bronchiectasis)
—— jacket, 204
—— kidney disease in, 203
—— leading to cardiac dilatation, 226
—— —— to septic meningitis, 298
—— lips in, 10
—— lobar, 207
—— —— abortive type of, 209
—— —— apical type of, 209
—— —— breathing in, 208
—— —— clinical picture in, 208
—— —— complications of, 209
—— —— cough in, 208
—— —— etiology of, 207
—— —— meningism in, 209
—— —— pathology of, 207
—— —— physical signs in, 208
—— —— pneumococcal septic æmia and, 207
—— —— prognosis of, 210
—— —— stages of, 207
—— —— symptoms of, 208
—— —— temperature in, 207,208
—— —— —— falling by crisis in, 207
—— —— treatment of, 210
—— mortality of, 33
—— nose in, 9
—— pneumococcal, 203
—— pulse-rate in, 225
—— rickets in, 203
—— right-sided, differential diagnosis of appendicitis from, 173
—— seasonal incidence of, 198
—— streptococcal, 203
—— types of, 209

Pneumonia, unresolved, 400
—— —— leading to bronchiectasis, 214
—— white, due to syphilis, 482
Pneumoradiograms, 332
Polio-encephalitis, 304, 307
—— differentiated from tuberculous meningitis, 291
—— treatment of, 307
Poliomyelitis, acute anterior, 300
—— —— —— age- and seasonal incidence of,300
—— —— —— cerebro - spinal fluid in, 304
—— —— —— clinical picture in, 302
—— —— —— diagnosis of, 304
—— —— —— epidemics of, 303
—— —— —— —— death - rate in, 305
—— —— —— etiology of, 300
—— —— —— incidence of cases in, 301
—— —— —— incubation period of, 300
—— —— —— mode of spread of infection in, 301
—— —— —— muscles in, 302
—— —— —— pathology of, 301
—— —— —— prognosis of, 304
—— —— —— treatment of, 305
—— —— —— types of, abortive, 303
—— —— —— —— ascending and descending, 304
—— —— —— —— bulbar and pontine, 304
—— —— —— —— cerebral and cerebellar, 304
—— —— —— —— meningitic, 304
—— —— —— —— neuritic, 303
—— —— —— —— spinal, 302
—— as cause of convulsions, 342
—— —— of spinal curvature, 364
—— cerebro-spinal fluid in, 295
—— chronic anterior, 308
—— differentiated from tuberculous meningitis, 291
—— length of life in, 295
Poliomyelitis, milk and, 63
—— polio-encephalitis and, 307
—— symptoms of, analysis of, 295
Polyneuritis, toxic, complicating typhoid fever, 410
Postbasic meningitis (*see* Meningitis, cerebro-spinal)
Postural albuminuria (*see* Albuminuria, orthostatic)
Posture, faulty, 363
Post-diphtheritic paralysis, 14, 312, 404
Post-encephalitis lethargica, 285
Post-vaccinal encephalitis, 415
Potassium bromide, rectal injection of, after tonsillectomy, 153
Potato-cream, 109
Poultices, starch, 386
Præcordial thrill in mitral stenosis, 12, 228, 452
Precocious puberty, 27
Pregnancy, breast-feeding in, 71
—— weaning in, 71
Premature birth, causes of, 53
—— infant, anæmia in, 61
—— —— bathing of, 55
—— —— "blue-turns" in, 60
—— —— caloric requirements of, 55
—— —— care of, 54
—— —— cerebral hæmorrhage in, 42
—— —— feeding of, 55
—— —— —— artificial, 57
—— —— —— —— times of, 59
—— —— —— breast-, 56
—— —— —— —— times of, 57
—— —— hospital treatment unnecessary for, 55
—— —— liability of, to von Jaksch's anæmia, 236, 238
—— —— maintenance of body-temperature of, 54
—— —— rickets in,prevention of, 60
—— tube feeder, 59
Prematurity preventing breast-feeding, 74
"Pre-rheumatism," 129
"Pre-tuberculosis," 129
Progressive spinal muscular atrophy (*see* Werdnig-Hoffmann paralysis)

Proptosis, 146
Protein content of milk, breast-,70
—— —— —— condensed, 65
—— —— —— cow's, 70
—— —— —— dried, full-cream, 66
—— —— —— —— humanized, 67
—— —— —— —— low-fat, 66
—— —— of patent starchy foods, 67
Protein-shock in asthma, 224
—— therapy in hæmophilia, 250
—— —— in rheumatoid arthritis, 461
Pseudo-hypertrophic muscular paralysis, 33, 316
Pseudo-paralysis, Parrot's, 480
Psoriasis, 388
—— treatment of, 388
Ptosis of eyelid, 9
—— of liver, 13
Puberty, enlarged thyroid preceding, 23
—— precocious, 27
Public School Anti-catarrh Vaccine, 413
Puerperal septicæmia, breast-feeding in, 71
Puffed rice, 134
"Puffing attacks," following epidemic encephalitis, 286
Pulmonary stenosis, 229
—— tuberculosis (*see* Tuberculosis, pulmonary)
Pulse-rate in infancy and childhood, 225
Puncture, cistern (*see* Cistern puncture)
—— lumbar (*see* Lumbar puncture)
Purpura, 247
—— age-incidence of, 247
—— blood-platelet count in, 248
—— coagulation time in, 248
—— due to scurvy, 108, 109
—— etiology and pathology of, 248
—— forms of, 247
—— hæmaturia in, 255
—— hæmorrhagica, 247
—— —— and hæmophilia, differences between, 249
—— —— and scurvy, 109
Purpura, Henoch's, 247
—— —— differentiated from intussusception, 181
—— —— mistaken for acute appendicitis, 247
—— in cerebro-spinal meningitis, 293
—— prognosis of, 248
—— rheumatic, 448, 451
—— simple, 247
—— symptoms and diagnosis of, 247
—— thrombocytopenic, 248
—— —— blood-platelet, count in, 248
—— treatment of, 248
Pyelitis, 256
—— accompanying parenchymatous nephritis, 277
—— age-incidence of, 256
—— as cause of anorexia, 355
—— —— of hæmaturia, 254
—— differentiated from miliary tuberculosis, 425
—— etiology of, 256
—— infective organisms in, 258
—— leading to interstitial nephritis, 279
—— mode of infection in, 256
—— pathology of, 257
—— prognosis of, 259
—— sex-incidence of, 256
—— simulating appendicitis, 174
—— symptoms and diagnosis of, 258
—— temperature in, 258
—— treatment of, 259
—— urine in, 258
Pyelocystitis, 262
Pyelonephritis, 257, 260
—— following pyelitis, 259
—— leading to interstitial nephritis, 279
Pyknolepsy, 346
—— as cause of convulsions, 342
Pyloric stenosis, congenital, 50,163
—— —— —— age- and sex-incidence of, 163
—— —— —— clinical picture in, 169
—— —— —— constipation in, 164
—— —— —— diagnosis of, 164
—— —— —— diarrhœa in, 166

Pyloric stenosis, congenital, pathology of, 163
—— —— —— peristaltic waves in, 164
—— —— —— pneumonia in, 167
—— —— —— post-operative feeding in, 167
—— —— —— pyloric tumour in, 164
—— —— —— symptoms of, 164
—— —— —— treatment of, 166
—— —— —— vomiting in, 163, 164
Pylorospasm and pyloric stenosis, 164, 165
Pyonephrosis, 261
Pyopericardium accompanying empyema, 214
Pyorrhœa, 156
Pyuria, chronic, resulting from pyelitis, 259

Q

Quarantine, observance of, results of, 389
—— periods for infectious diseases, 390
Quinine in treatment of rumination, 142
Quinsy, 150

R

Radiomalt, 106, 357
Radiostol, 104, 357
Radiostoleum, 106
Radium treatment in laryngeal papilloma, 194
—— —— in lymphadenoma, 247
Rammstedt's operation, 167
Rash, belladonna, 384
—— bromide, 383
—— enema, 384
—— heat, 382
—— of chicken-pox, 402
—— of congenital syphilis, 479
—— of measles, 395
—— of pink disease, 310
—— of rheumatism, 451
—— of rubella, 394
—— of scarlet fever, 391
—— of typhoid fever, 406
—— of urticaria, 382
—— serum, 384
Rashes, drug, 383
Rashes, napkin, 372
Recognition of persons, 14
Recolac dried milk, 67, 85, 88
Rectal injection of paraldehyde for anæsthesia, 153
—— —— of potassium bromide after tonsillectomy, 153
—— saline in dietetic diarrhœa, 123, 127
—— —— in pyloric stenosis, 169
Rectum, prolapse of, 186
—— —— treatment of, 187
Red Butterfly condensed milk, composition of, 65
Redux crepitations in pneumonia, 209
Regal condensed milk, composition of, 65
Renal dwarfism, 27, 279, 280, 281
—— function tests, 252
—— sarcoma, 430
Respiration rate in infancy and childhood, 225
Respiratory diseases, 191
—— —— mortality of, in childhood, 33
—— tract, upper, infections of (*see* Nose and throat infections)
Rest, importance of, in abdominal tuberculosis, 430
—— —— in chorea, 447
—— —— in nervous exhaustion, 25, 353
—— —— in rheumatic carditis, 455
Restlessness due to underfeeding, 75
Retropharyngeal abscess, 151
Rhagades, 10, 479
Rheumatic carditis, 227, 441, 444, 448, 452
—— —— accompanying chorea, 446
—— —— care of child in, 454
—— —— complicating scarlet fever, 392
—— —— convalescence in, 455
—— —— following scarlet fever, 443
—— —— hypertrophy of heart in, 227, 454

Rheumatic carditis, mitral regurgitation in, 452
—— —— —— stenosis in, 452
—— —— murmurs in, 227, 452
—— —— pathology of, 443
—— —— posture in, 455
—— —— prognosis of, 458
—— —— pulse-rate in, 226, 453, 455, 456, 457
—— —— sex-incidence of, 442
—— —— temperature in, 456,457
—— —— treatment of, 455
—— —— valves in, 227, 452, 453
—— —— (*see also* Rheumatic pericarditis; Pericardium, adherent)
—— encephalitis (*see* Chorea)
—— endocarditis, 444
—— erythema, 448, 451
—— fever, mortality of, 33, 34,
—— —— (*see* also Rheumatism)
—— fibrosis, 444
—— heart disease (*see* Rheumatic carditis)
—— manifestations, various, 448
—— myositis, 444, 448
—— nodules, 441, 444, 448, 450
—— pericarditis, 228, 444, 453
—— purpura, 448, 451
—— tachycardia, 226
Rheumatism, 441
—— acute, differentiated from rheumatoid arthritis, 460
—— age-incidence of, 442
—— anæmia in, 449, 458
—— appendicitis and, 172
—— arthritis in, 441, 448, 450
—— as cause of anorexia, 355
—— —— of wasting, 24
—— cardiac murmurs in, 227, 228, 452
—— clinical picture in, 448
—— complicating nose and throat infections, 151
—— dampness and, 442, 458
—— differentiated from osteomyelitis, 371
—— —— from poliomyelitis, 304
—— environment and, 442
—— etiology of, 441
—— geographical incidence of, 443
—— heredity and, 442
—— initial attack in, 442
Rheumatism, involvement of heart in, 226, 443, 452
—— muscular, 444, 448
—— of nervous system (*see* Chorea)
—— of subcutaneous tissues, 448
—— of throat, 448
—— organisms of, 441
—— pains due to, 449
—— prevention of, 458
—— prognosis of, 458
—— rash of, 451
—— scarlet fever and, 443
—— seasonal variation in, 442
—— sex-incidence of, 441
—— skin manifestations of, 448, 451
—— strata of society affected by, 443
—— symptoms of, 449
—— tonsil as portal of infection in, 151, 441
—— tonsillitis in, 448, 451
—— weather and, 443
—— (*see also* Rheumatic; Rheumatoid arthritis)
Rheumatoid arthritis, 459
—— —— differential diagnosis of, 460
—— —— etiology of, 459
—— —— pathology of, 459
—— —— treatment of, 461
—— —— Wassermann reaction and, 460
Ribs, beading of, 12, 100, 103, 108
—— examination of, 11, 12
—— greenstick fracture of, 12
Rice flour, 89
Rickets, 99
—— age-incidence of, 99
—— anæmia in, 100
—— and calcium deficiency, 61, 102, 103, 350
—— and tendency to bronchitis, 195
—— —— to umbilical hernia, 190
—— arms in, 99
—— artificial sunlight treatment in, 28
—— as cause of anorexia, 355
—— —— of convulsions, 102, 347, 350
—— —— of knock-knee and flat foot, 362

Rickets as cause of spinal curvature, 364
—— bones in, 99, 103
—— carpopedal spasm in, 101
—— cartilaginous enlargements in, 103
—— chest in, 12, 99
—— Chvostek's sign in, 10, 101
—— clinical picture of, 99
—— " congenital," 368
—— convulsions in, 102, 347, 350
—— cramps in, 102
—— craniotabes in, 101
—— crowing in, 102
—— dentition in, 29, 31, 99
—— diagnosis of, 103
—— diet in, 104
—— due to interstitial nephritis, 280
—— due to over-administration of thyroid, 338
—— dwarfing in, 104
—— epiphyses in, 103
—— etiology of, 102
—— facial irritability in, 10, 101
—— fatal terminations of, 104
—— hair in, 8
—— hands in, 101
—— head in, 8, 99, 101
—— in premature infants, 61
—— influence of on infections, 104
—— laryngismus stridulus in, 102
—— leading to broncho-pneumonia, 200
—— legs in, 13, 99, 101
—— muscles in, 101
—— nervous, 101, 350
—— nystagmoid movements in, 9
—— pathology of, 103
—— prognosis of, 104
—— ribs in, 12, 100, 103, 108
—— signs and symptoms of, 100
—— stomach in, 101
—— stunting in, 27
—— sunshine and, 28, 103, 105
—— sweating of head in, 100
—— teeth in, 29, 99
—— tetany in, 101, 104
—— toes in, 102
—— treatment of, 104
—— Trousseau's sign in, 101
—— vitamin deficiency and, 102, 103, 106
—— walking capacity and, 15
Rickets, X-ray examination in, 104
Ridge's Food, composition of, 68
Ringworm, of body, 377
—— of nails, 378
—— of scalp, 377
—— —— treatment of, 377
Robb's Biscuit, 79, 89, 91
Robinson's Patent Barley, 69, 89, 90
—— —— Groats, 69, 89, 90, 142, 180
Roller process of drying milk, 64
Rose spots in typhoid fever, 406
Rotation of breasts in feeding, 77
Rothera's test, 252, 467
Round-shouldered child, 363
Roundworm infection, 475
Rubella, 393
—— age-incidence of, 393
—— clinical picture and symptoms of, 394
—— incubation period of, 393
—— period of infectivity in, 390
—— quarantine period and rules to be observed in, 390
—— rash of, 394
—— return to school after exposure to infection of, 390
—— treatment of, 394
Rumination, 140
—— diagnosis of, 141
—— etiology of, 140
—— feeding in, 67
—— treatment of, 141
Ryvita Crispbread, 69, 131

S

St. Margaret's Hospital for ophthalmia neonatorum, 39
St. Vitus's dance (*see* Chorea)
Salicylates, use of, in epidemic encephalitis, 288
Saline, half-strength normal, preparation of, 127
—— —— —— use of, 59, 123, 127
—— in acute hæmorrhagic nephritis, 273
—— in congenital pyloric stenosis, 166, 167, 168, 169
—— in dietetic diarrhœa, 123, 126, 127
—— in status epilepticus, 346
—— normal, preparation of, 126

Saline, normal, use of, 126
Saliva, excess of, causing bronchitis, 195
Salts in milk, 70, 71
Salvitæ, 130, 134, 140
Savory & Moore's Food, 69, 123, 125, 142, 205
Scabies, 375
Scalp, examination of, 8
—— hæmatoma of, 8
Scarlatina, 391
Scarlet fever, 390
—— —— accompanied by nose and throat infections, 150
—— —— age- and seasonal incidence of, 391
—— —— "carriers," 391
—— —— clinical picture in, 391
—— —— complications of, 146, 392
—— —— contacts, protection of, 393
—— —— desquamation in, 392
—— —— diagnosis of, 391
—— —— Dick test in, 393
—— —— etiology of, 391
—— —— followed by nephritis, 271, 274, 279
—— —— incubation period of, 390
—— —— influence of on tuberculosis, 424
—— —— leading to rheumatism, 443
—— —— milk and, 63
—— —— mode of infection in, 390
—— —— mortality of, 33, 389
—— —— prognosis of, 392
—— —— quarantine period and rules to be observed in, 390
—— —— rash of, 391
—— —— susceptibles, immunization of, 393
—— —— symptoms of, 391
—— —— tongue in, 392
—— —— treatment of, 392
—— —— —— prophylactic, 393
Schick test, 405
School and nervous exhaustion, 25
—— attendance and infectious diseases, 389, 390
School (*see also* Kindergarten)
Schultz-Charlton test, 391
"Scissor-leg" attitude in spastic paralysis, 43
Scoliosis, 364
—— significance of, 12
Scott's Emulsion, 357
—— Oat Flour, composition of, 69
Screaming attacks in congenital heart disease, 232
Scurf of scalp, 379
Scurvy, infantile, 106
—— —— age-incidence of, 106
—— —— artificial feeding and, 106
—— —— as cause of protrusion of eyeball, 146
—— —— clinical picture in, 109
—— —— diagnosis of, 108
—— —— —— from poliomyelitis, 304
—— —— diet in, 109
—— —— etiology of, 107
—— —— gums in, 10, 108
—— —— hæmorrhage in, 107
—— —— pathology of, 107
—— —— prognosis of, 109
—— —— prophylaxis in, 109
—— —— purpura in, 108
—— —— ribs in, 108
—— —— sternum in, 12, 108
—— —— symptoms of, 107
—— —— treatment of, 109
—— —— urine in, 108
—— —— vitamins and, 105, 107, 109
Sea air, best in abdominal tuberculosis, 432
Seborrhœa, 379
—— treatment of, 379
Secway dried milk, composition of, 66
Semprolin, 136, 137, 139
Senses, special, development of, 14
Sepsis neonatorum, 36
—— umbilical, 13, 36
Septicæmia accompanying empyema, 214
—— chronic, differentiated from lymphadenoma, 246
—— resulting from umbilical sepsis, 13
Serum, antidiphtheritic, 193

Serum, antistreptococcal, 37
—— antitetanic, 38
—— convalescent, in treatment of acute anterior poliomyelitis, 306
—— rash, 384
—— sickness, 297
—— therapy in cerebro-spinal meningitis, 296
—— —— in diphtheria, 404, 405
—— —— in hæmophilia, 250
—— —— in infective diarrhœa, 119
—— —— in measles, 396
—— —— in sepsis neonatorum, 36, 37
—— —— in tetanus neonatorum, 38
Shiga dysentery, 116, 118
Shingles (*see* Herpes zoster)
Shoes, fitting of, 17
—— for flat feet, 17
Shredded Wheat, 134
Sinus infections, leading to septic meningitis, 298
Sister Laura's Food, 69
Sit up, ability to, 15
Sitz baths in treatment of masturbation, 359
Sixth nerve, maldevelopment of, as cause of squint, 144
Skin diseases, 372
—— infections due to umbilical sepsis, 37
—— tuberculosis of, 418
Skin-tests in asthma, 223
Skull, bossing of, due to syphilis, 484, 485
—— examination of, 7
—— in rickets, 8, 101
Sleep, abnormalities in, causes of, 27
—— disturbed, causes of, 27, 357
—— requirements of normal child, 27
Sleeplessness due to underfeeding, 75
" Sleepy sickness," 283
S.M.A. dried milk, composition of, 67
Smallpox prophylaxis, vaccination in, 413
Snuffles, due to syphilis, 479
Solid food, introduction of, 93
Somnolence, diurnal, 28, 286
Sonne bacilli in stools, 118
" Soothing powders " as cause of bromide rash, 383
Sore nipples, 74
Southey's tubes, 277
Soxhlet apparatus, 88
—— feeding bottle, 87, 88
Spasmophilia, 101, 339, 340, 350
Spastic diplegia (*see* Spastic paralysis)
—— paralysis, 40, 322
—— —— and status epilepticus, 346
—— —— etiology of, 41
—— —— pathology of, 42
—— —— prognosis of, 44
—— —— symptoms and diagnosis of, 42
—— —— treatment of, 44
Special senses, development of, 14
Speech, development of, 14
Sphagnum moss in treatment of enuresis, 265
Spina bifida and enuresis, 262
Spinal caries, 364, 437
—— —— as cause of spinal curvature, 364
—— curvature, causes of, 364
—— —— treatment of, 364
—— tuberculosis, 437
—— —— as cause of spinal curvature, 364
Spine, examination of, 12
Spirochæte pallida, 478
Spleen, palpable, significance of, 13
Splenectomy in acholuric family jaundice, 236
—— in Banti's disease, 233
—— in lymphadenoma, 247
—— in thrombocytopenic purpura, 248
—— in von Jaksch's anæmia, 238
Splenic anæmia (*see* Anæmia, splenic)
—— enlargement in Banti's disease, 233
—— —— in glandular fever, 154
—— —— in lymphadenoma, 243
—— —— in spleno - medullary leukæmia, 241

Splenic enlargement in von Jaksch's anæmia, 236
Spleno-medullary leukæmia (*see* Leukæmia, spleno-medullary)
Splint, Fairbank, 48
—— for head in cervical adenitis, 434
—— Liston's, 187
Splints, cardboard, 205, 386
—— celluloid, 306
—— night, 363
Spoon-feeding, commencement of, 89
Spotted fever (*see* Meningitis, cerebro-spinal)
Spray process of drying milk, 64
Sprue, adult, and cœliac disease, 113
Squint, causes of, 9, 144
—— concomitant, convergent, 144
—— —— divergent, 144
—— functional, 144
—— —— etiology of, 144
—— —— treatment of, 144
—— in diphtheria, 404
—— in epidemic encephalitis, 285
—— organic, 144
—— —— causes of, 144
Stammering, 361
Stand up, ability to, 15
Staphylococcal vaccine in blepharitis and styes, 145
—— —— in boils, 379
Starch, addition of, to food in rumination, 142
—— dyspepsia (*see* Dyspepsia, starch)
—— in infant feeding, 83, 89
—— poultices, method of making, 386
Starchy patent foods, 67
"Start-Rite" shoes, 17, 363
Static curves, 364
Status epilepticus, 343, 346
—— lymphaticus and enlarged thymus, 218
Steam-kettle in bronchitis, 197
—— in croup, 192
Steam-tent in broncho-pneumonia, 206
—— not to be used in bronchitis, 197
Stenosis, duodenal (*see* Duodenal stenosis)
—— œsophageal, 162
—— pulmonary, 229
—— pyloric (*see* Pyloric stenosis)
Sternal depression, 12, 108
—— dislocation in scurvy, 12, 108
Sternomastoid, hæmatoma of, 46
Still-birth due to syphilis, 479
Still's disease (*see* Rheumatoid arthritis)
Stimulation of lactation by suction, 75
Stomach, obstruction of, as cause of vomiting, 358
Stomatitis, aphthous, 156
—— complicating measles, 395
—— —— throat infections, 151
—— tonsillar infections and, 151
—— ulcerative, 156
—— —— clinical picture in, 156
—— —— complicating scarlet fever, 392
—— —— diet in, 158
—— —— etiology and pathology of, 156
—— —— gums in, 10
—— —— treatment of, 157
—— (*see also* Thrush)
Stools, abnormal, appearance of, 96
—— accompanying hæmorrhoids or anal fissure, 97
—— —— intussusception, 97
—— acid, 80, 82, 96, 99
—— alkaline, 80, 96, 99
—— blood in, 97
—— blood-tinged in infective diarrhœa, 118
—— brown, 96
—— curds in, 82, 97
—— dysentery organisms in, 118
—— fat in, 96, 113
—— following administration of castor oil, 97
—— green, causes of, 96, 97
—— grey, causes of, 96
—— "hunger," 75
—— in absence of bile-ducts, 96
—— in acute infections of bowels, 97
—— in catarrh of bowel, 97
—— in cœliac disease, 96, 113
—— in colitis, 97

Stools in congenital pyloric stenosis, 97
—— in dietetic diarrhœa, 120, 121, 122
—— in enteritis, 97
—— in infective diarrhœa, 118
—— in melæna neonatorum, 97
—— in mucous colitis, 179
—— in overfeeding on fats or carbohydrates, 97
—— in starvation, 97
—— in typhoid-dysentery infections, 97
—— Morgan bacilli in, 118
—— mucus in, 97
—— mustard-coloured, 96
—— normal, colour of, 96
—— —— composition of, 96
—— —— reaction of, 96, 99
—— odour of, offensive, 96
—— of artificially-fed infants, 96, 99
—— of breast-fed infants, 96, 99
—— of new-born infants, 96
—— pale-coloured, 110
—— paratyphoid organisms in, 118
—— "pea-soup," 406
—— pus in, 97
—— putty-like in cœliac disease, 110
—— "red-currant jelly," 97
—— Sonne bacilli in, 118
—— tarry, 97
—— treatment of, for examination, 96
—— unusual ingredients of, 97
—— white, causes of, 96
—— worms in, 473
"Strawberry tongue," 392
Streptococcal meningitis (*see* Meningitis, septic)
Streptococcus antitoxin (scarlatina) serum, 454
Stricture, œsophageal, 162
Stridor, congenital laryngeal (*see* Laryngeal stridor, congenital)
"Strumous" child and conjunctivitis, 145
Stunting due to rickets, 104
Stuttering, 361
Styes, 145
Sub-diaphragmatic effusion, 13
Sucking of fingers as cause of vomiting, 358
Sucking-pads, 10
Sucrose in condensed milk, 63
Sugar in artificial feeding, 82, 85
—— in breast-milk, 70
—— in urine in diabetes, 463
—— —— in renal glycosuria, 464
—— —— test for, 252, 467
—— of cow's milk, 70, 82
Summer diarrhœa, 116
Sunlight, artificial, after influenza, etc., 29
—— —— in asthma, 28
—— —— in cancrum oris, 158
—— —— in cœliac disease, 114
—— —— in general debility, 138
—— —— in nephritis, 277, 282
—— —— in pink disease, 312
—— —— in pseudo-hypertrophic muscular paralysis, 318
—— —— in rickets, 28, 105, 350, 362
—— —— in spasmophilia, 350
—— —— in tuberculosis, 423, 434
—— —— value of, 28
Sunshine, artificial and natural, value of, 28, 29
—— benefits of, 28, 29, 105, 114, 131, 137, 138, 238, 277, 282, 312, 350, 362, 423, 431, 434
—— deprivation of, and rickets, 103
Supplementary feeding, 77
Suprarenal, tumour of, 27
Surgical diseases, mortality of, 34
Sweating of head in rickets, 100
Symptomatic diarrhœa (*see* Diarrhœa, symptomatic)
Syphilis, acquired, 478
—— congenital, 372, 478
—— —— as cause of wasting, 24
—— —— bossing of skull in, 8, 481, 484
—— —— bones in, 480
—— —— breast feeding in, 71
—— —— cirrhosis of liver due to, 38, 482, 484
—— —— clinical picture in, 479
—— —— differentiation of lymphadenoma from, 246
—— —— enlargement of liver due to, 482

Syphilis, congenital, enlargement of spleen due to, 480, 481
—— —— eye lesions due to, 482
—— —— head in, 8, 481, 484
—— —— leading to fibrosed kidney, 279
—— —— lips in, 10
—— —— macerated fœtus due to, 479
—— —— mental deficiency due to, 482
—— —— nose in, 9
—— —— of bones, 480
—— —— organism of, 478
—— —— paroxysmal hæmoglobinuria due to, 483
—— —— pathology of, 484
—— —— peg-shaped teeth due to, 483
—— —— prognosis of, 487
—— —— pseudo-paralysis due to, 480
—— —— rash in, 372, 479
—— —— snuffles as a sign in, 479
—— —— still-birth due to, 479
—— —— summary of manifestations of, 483
—— —— treatment of, 484
—— —— —— arsenic injections in, 486
—— —— —— mercury in, 487
—— —— —— prophylactic, 487
—— —— Wassermann reaction in, 478, 481
—— extra-genital, 478
—— infection in, 478
—— infectiousness of, 478
Syphilitic arthritis, 460
—— carditis, 482
—— condylomata, 479, 480
—— dactylitis, 481
—— epiphysitis, 480
—— laryngitis, 480
—— meningitis, 481
—— nephritis, 351, 482, 483
—— osteitis, 481
—— pemphigus, 479
—— perforation of soft palate, 480, 481
—— periostitis, 481
—— rhagades, 10, 479, 480
—— testis, 482
—— wig, 479

T

Tabes mesenterica, 426, 427, 429, 432
—— —— diagnosis of, 429
—— —— prognosis of, 432
—— —— symptoms of, 428
—— —— treatment of, 430
Tache cérébrale in tuberculous meningitis, 291
Tachycardia, 225
—— accompanying habit spasm, 354
—— in hyperthyroidism, 22, 23
—— in miliary tuberculosis, 225
—— in pink disease, 225, 310
—— nervous, 225
—— paroxysmal, 226
—— rheumatic, 226
Tænia cucumerina, 475
—— mediocanellata, 472
Tapeworm infection, 472
—— —— symptoms and diagnosis of, 473
—— —— treatment of, 473
Taxol, 139
Tay-Sachs' disease, 48
Teeth, care of, 161
—— caries in (*see* Dental caries)
—— cleaning of, 161
—— cutting of (*see* Dentition)
—— septic, removal of, in catarrhal jaundice, 184
Teething, as cause of disturbed sleep, 357
—— —— of frequency of micturition, 267
—— breath-sounds in, 12
—— scurvy and, 109
—— spots, 383
Temperature, body-, maintenance of in premature infant, 54
Test-feeds, 76, 77, 79
—— in aerophagia, 143
Testes, undescended, 13
Testis, syphilitic, 482
Tetanus neonatorum, 38
Tetany, acute, in cœliac disease, 110
—— (spasmophilia) due to rickets, 101
Thallium acetate in treatment of ringworm, 377
Thin child, 23, 129
Thinness, constitutional, 24, 129

Thinness, constitutional, treatment of, 25
—— diseases causing, 24
Thirst, due to diabetes, 462
Threadworm infection, 475
—— —— symptoms of, 476
—— —— treatment of, 477
Throat and nose infections (*see* Nose and throat infections)
—— diseases of, 144
—— examination of, 10
—— painting of, 152
—— soreness of, due to rheumatism, 450
—— swabs in diphtheria, 403
—— —— in post-diphtheritic paralysis, 314
—— —— in spasmodic croup, 192
Thrombocytopenic purpura (*see* Purpura, thrombocytopenic)
Thrush, 155
Thumb-sucking, 360
Thymic asthma, 218
Thymus, enlarged, 218
Thyroid gland, adenoma of, 338
—— —— and cretinism, 334
—— —— congenital absence of, 26
—— —— enlargement of, 23
—— —— in treatment of cretinism, 336
—— —— —— of obesity, 22
Tibiæ, sabre-shaped, due to syphilis, 484
Time required for breast-feed, 73
Times of feeding, 73
Tinea capitis, 377
—— circinata, 377
—— corporis, 377
—— unguium, 378
Tip-top condensed milk, composition of, 65
Tongue, dirty, due to dental caries, 160
—— geographical, 159
—— —— treatment of, 159
—— in mongolism, 323
—— in thrush, 155
—— "strawberry," 392
Tonsillar glands, enlarged, due to tuberculosis, 433
Tonsillitis, 151
Tonsillitis, accompanied by herpes febrilis, 381
—— accompanying otitis media, 147
—— and rheumatism, 448, 451
—— as cause of acidosis, 132
—— —— of mucous colitis, 179
—— complicating chicken-pox, 403
—— follicular, differentiated from diphtheria, 403
—— leading to septic meningitis, 298
—— mortality from, 33
—— with umbilical colic, appendicitis and, 174, 175
Tonsils and immunity, 153
—— as portal for rheumatic infection, 151, 451
—— examination of, 11
—— function of, 153
—— removal of, 152
—— —— after-treatment in, 153
—— —— anæsthesia for, 153
—— —— in asthma, 221
—— —— in catarrhal jaundice, 184
—— —— in chorea, 448
—— —— in nephritis, 272
—— —— in rheumatoid arthritis, 461
—— septic, and conjunctivitis, 145
—— —— and dental caries, 160
—— —— and nephritis, 151
—— —— and stomatitis, 151
—— tuberculous infection of, 154
Torticollis, 11, 46
Toxoid antitoxin serum in diphtheria, 405
Tracheitis complicating chicken-pox, 402
Tracheotomy in croup, 192, 193
—— in laryngeal papilloma, 194
Train-sickness, 132, 219
Training of child in good habits of micturition and defæcation, 15
Transfusion of blood (*see* Blood transfusion)
Transillumination in mastoid disease, 149
"Trench mouth," 156
Trismus neonatorum, 38
Trousseau's sign in rickets, 101

Trufood, full-cream dried milk, composition of, 66
—— humanized, dried milk, 67, 85
—— skimmed dried milk, composition of, 66
Trumilk, composition of, 66]
Trusses for hernia, 189
Tube feeding, 45, 59, 60, 287, 313
Tubercle bacilli, human and bovine, 417
Tuberculides, 418
Tuberculin dilutions, 438
—— test in asthma, 221
—— —— in cervical adenitis, 434
—— —— in erythema nodosum, 382
—— —— intracutaneous, 419, 434, 438
—— —— —— method of making up solution for, 438
—— —— —— negative, 440
—— —— —— positive, 439, 440
—— —— —— technique of, 438
—— —— —— when to use, 439
—— —— subcutaneous, 419
—— treatment contra-indicated in pulmonary tuberculosis, 423
—— —— —— in tuberculous cervical adenitis, 434
Tuberculin-tested milk, 63
Tuberculomata, 290
Tuberculosis, 417
—— abdominal, 418, 424, 426
—— —— age-incidence of, 426
—— —— bone tuberculosis secondary to, 436
—— —— chronic diarrhœa in, 429
—— —— diagnosis of, 429
—— —— diet in, 431
—— —— differentiated from intussusception, 181
—— —— etiology of, 426
—— —— pain in, 428
—— —— pathology of, 427
—— —— perforation of ulcer in, 428
—— —— physical findings in, 428
—— —— prognosis of, 432
—— —— simulating appendicitis, 175
—— —— symptoms of, 428
Tuberculosis, abdominal, treatment of, 430
—— —— ulceration of bowel in, 426, 427, 428, 429, 432
—— —— (*see also* Tabes mesenterica; Tuberculous peritonitis; Tuberculous ulceration of bowel)
—— administration of tuberculin in, 419, 423, 434
—— and bronchiectasis, 216
—— as cause of anorexia, 355
—— —— of wasting, 24
—— bacillus, infection with, 417
—— bone, 436
—— —— clinical picture in, 437
—— —— etiology and pathology of, 436
—— —— primary focus in, 436
—— —— treatment of, 437
—— breast-feeding in, 71
—— diagnosis of, 419
—— etiology of, 417
—— general remarks on, 417
—— genito-urinary, 420
—— hilum, 421
—— immunity in, 419
—— infection of bloodstream in, 419
—— mediastinal gland, 421
—— —— bone tuberculosis secondary to, 436
—— —— diagnosis of, 422
—— —— prognosis, 422
—— miliary, 418, 419, 423
—— —— age-incidence of, 424
—— —— clinical picture in, 424
—— —— complicating whooping-cough, 400
—— —— diagnosis of, 425
—— —— differentiated from lymphadenoma, 246
—— —— etiology and pathology of, 423
—— —— infection of lungs in, 424
—— —— —— of meninges in, 423, 425
—— —— prevention of, 425
—— —— primary focus in, 423
—— —— prognosis and treatment of, 425

Tuberculosis, miliary, pulse-rate in, 225
—— —— seasonal incidence of, 424
—— —— tuberculous meningitis and, 288, 423
—— milk and, 417
—— modes of infection in, 417
—— mortality of, 33, 34
—— of alimentary tract, 418
—— of ankle, 437
—— of bones and joints, 418
—— —— —— (*see also* Tuberculosis, bone)
—— of brain and meninges, 418
—— —— —— (*see also* Meningitis, tuberculous)
—— of glands, 418, 421, 426, 433
—— —— diagnosis of lymphadenoma from, 246
—— —— (*see also* Tuberculous cervical adenitis)
—— of hip, 437
—— of kidney, 418
—— of knee, 437
—— of lung, 418
—— of skin, 418
—— of spine, 437
—— prevention of, 420
—— prognosis, 419
—— pulmonary, 418, 420
—— —— adult type of, 421, 422
—— —— clinical picture in, 421
—— —— cough in, 421
—— —— diagnosis of, 422
—— —— etiology and pathology of, 420
—— —— in childhood, 417
—— —— initial lesion in, 420
—— —— prognosis of, 422
—— —— symptoms of, 421
—— —— treatment of, 420, 423
—— sites of, in body, 418
—— spleen in, 13
—— tonsils and, 154
—— tuberculin tests in, 419, 438
—— X-ray examination in, 419
Tuberculous bacillæmia, erythema nodosum and, 381
—— cervical abscess, 433
—— —— adenitis, 433
—— —— —— clinical picture in, 433
Tuberculous cervical adenitis, etiology and pathology of, 433
—— —— —— treatment of, 433
—— dactylitis, 437
—— hip, 437
—— infection of brain and meninges, 418
—— meningitis (*see* Meningitis, tuberculous)
—— peritonitis, 426
—— —— ascitic form of, 426, 427, 428, 430
—— —— —— —— diagnosis of, 430
—— —— pathology of, 427
—— —— plastic form of, 426, 428, 429, 430
—— —— —— —— diagnosis of, 430
—— —— prognosis of, 432
—— —— symptoms of, 428, 430
—— —— treatment of, 431
—— pleurisy (*see* Pleurisy, tuberculous)
—— sinus, 433
—— ulceration of bowel, 424, 426
—— —— —— diagnosis of, 429
—— —— —— pathology of, 427
—— —— —— prognosis of, 432
—— —— —— symptoms of, 428
—— —— —— treatment of, 430
Tumour of brain, 11, 28, 48, 327
—— of liver, 13
—— of pineal gland, 28
—— of suprarenal, 27
—— ovarian, 27
—— pyloric, 164
Twins, liability of, to von Jaksch's anæmia, 237
Typhoid fever, 405
—— —— blood-count in, 406
—— —— "carriers," 406
—— —— clinical picture in, 406
—— —— complications of, 410
—— —— diagnosis of, 406
—— —— differentiated from lymphadenoma, 246
—— —— —— from miliary tuberculosis, 425
—— —— etiology of, 405
—— —— headache in, 406, 407

Typhoid fever, leading to cardiac dilatation, 226
—— —— mode of infection in, 405
—— —— mortality of, 407
—— —— pathology of, 407
—— —— prognosis of, 407
—— —— rash of, 406
—— —— resemblance of pneumococcal peritonitis to, 177
—— —— "rose spots" in, 406
—— —— spleen in, 406
—— —— stools in, 406
—— —— treatment of, 408
—— —— wasting in, 407
—— —— Widal test in, 406

U

Ulcer, corneal, 145, 380
—— duodenal, 171
—— meatal, 13, 267
Ulcerative stomatitis (*see* Stomatitis, ulcerative)
Ulcers, tuberculous, of bowel, 424, 426, 427, 428, 429, 432
Ultra-violet light treatment in impetigo contagiosa, 378
—— —— —— in lupus vulgaris, 380
—— —— —— in tuberculous cervical adenitis, 434
Umbilical colic and sore throat, 151
—— —— simulating appendicitis, 174, 175
—— hernia (*see* Hernia, umbilical)
—— infection, 13, 36
—— —— treatment of, 37
Umbilicus, condition of, in the new-born, 13
Underfeeding, as cause of constipation in infants, 136, 138
—— —— of disturbed sleep, 357
—— on breast, causes of, 75
—— —— diagnosis of, 76
—— —— due to congenital heart disease, 232
—— results of, 82
Undescended testes, 13
Unsweetened condensed milk mixture, 57, 64, 168
Uræmia, 259, 280, 282
Uræmic convulsions, 351
Urea, administration of, in nephritis, 277
Urea-concentration, normal value of, 252
—— test, 252
Urethritis, complicating vulvovaginitis, 270
Urinary antiseptics, 260, 262
—— tract, congenital malformations of, 255
—— —— infections of, 256
Urine, blood in (*see* Hæmaturia)
—— collection of specimen of, 251
—— examination of, 252
—— —— for acetone, 252, 467
—— —— for albumin, 252
—— —— for blood, 252
—— —— for diacetic acid, 252, 467
—— —— for globulin, 252
—— —— for pus, 252
—— —— for sugar, 252, 467
—— —— in diabetes, 467
—— —— starch powder leading to fallacy in, 252
—— glucose in, in diabetes, 463
—— normal quantity of, 251
—— reducing substance in, significance of, 463
—— smoky, in scurvy, 108
Urticaria, 382
—— and asthma, 219, 221
—— and chicken-pox, 383
—— causes of, 382
—— clinical picture in, 383
—— foods causing, 383
—— in dentition, 31
—— papulosa, 382
—— treatment of, 383

V

Vaccination, 413
—— a function of tonsil, 153
—— complicated by encephalitis, 415
—— technique of, 414
Vaccine therapy in influenza, 413
—— —— in rheumatoid arthritis, 461
—— —— in styes, 145

Vaccine therapy in treatment of boils, 379
—— —— in whooping-cough, 401
Valvular disease of heart, 227
Varicella (*see* Chicken-pox)
Vegetables in mixed feeding, 91, 92
Verrucæ, 379
Vertebræ, congenital malformations of, 12, 364
—— wedged, 364
Vigantol, 104, 106
Vincent's angina, 150, 156
—— spirochæte, 156, 158
Violence, mortality from, 33, 34
Viosterol, 104, 106
Virol and Milk, composition of, 66
Vitamalt, 106
Vitamin A, 105
—— —— foods containing, 105
—— —— in milk, 105
—— B, 105
—— —— and beriberi, 105
—— —— and pellagra, 105
—— —— foods containing, 105
—— C, 105
—— —— and scurvy, 106
—— —— foods containing, 106
—— D, 106
—— —— and rickets, 106
—— —— foods containing, 102, 106
—— —— —— deficient in, 102
Vitamins, absence of, a factor in anorexia, 355
—— and calcification of bones, 103
—— and dentition, 29
—— and rickets, 29, 102, 105
—— and scurvy, 106, 109
—— in milk, 63, 64, 87, 105, 106, 107
Vitmar, 105
Volvulus, 182
—— clinical picture in, 182
—— etiology of, 182
—— simulating appendicitis, 175
—— treatment of, 182
Vomiting, cyclical, 132, 180
—— —— acidosis and, 132
—— —— air-hunger and, 133
—— —— asthenia and, 133
—— —— cardio-vascular instability and, 133
—— —— diagnosis of, 133
Vomiting, cyclical, diet in, 133, 134
—— —— etiology of, 132
—— —— gastric attacks and, 133
—— —— hereditary tendencies in, 132
—— —— hypotonia and, 133
—— —— liver attacks and, 133
—— —— symptoms of, 133
—— —— treatment of, 133, 134
—— —— —— things to be avoided in, 135
—— during dentition, 30
—— in overfeeding, 78
—— in ulcerative stomatitis, 157
—— in underfeeding, 75
—— mechanical, 358
—— nervous, causes of, 358
—— projectile, in pyloric stenosis, 163, 164
Von Graefe's sign, 354
Von Jaksch's anæmia (*see* Anæmia, von Jaksch's)
Vulvo-vaginitis, 269
—— and pneumococcal peritonitis, 176
—— clinical picture in, 270
—— due to threadworm infection, 476
—— gonorrhœal, 269, 270
—— non-gonorrhœal, 269
—— organisms concerned in, 269
—— treatment of, 270

W

Waddling gait, 13
Wakefulness, nocturnal, 28, 286
Walking, progress in, 15
Wander's Nutromalt, 82, 85
Warts, 379
—— treatment of, 379
Wassermann reaction, epilepsy and, 342
—— —— hydrocephalus and, 332
—— —— in syphilis, 478, 481, 484, 487
—— —— interstitial nephritis and, 282
—— —— lymphadenoma and, 246
—— —— paroxysmal hæmoglobinuria and, 255
—— —— rheumatoid arthritis and, 460
—— test in adoption, 32

Wasting, causes of, 24
—— due to diabetes, 462
—— due to pulmonary tuberculosis, 421
—— due to whooping-cough, 400
—— in typhoid fever, 407
Water content of milk, breast-, 70
—— —— —— cow's, 70
—— —— —— dried, full-cream, 66
—— —— —— —— humanized, 67
—— —— —— —— low-fat, 66
—— in treatment of constipation, 139
—— on brain (*see* Hydrocephalus)
Water-soluble B vitamin, 105
—— C vitamin, 105, 106
Weaning, 71, 79
Weighing of infants, 18
Weight, failure to gain, due to heart disease, 232
—— growth in, 18
—— increase of, rate of, 18
—— —— (*see also* Obesity)
—— loss of, causes of, 18
—— —— (*see also* Wasting; Thinness)
—— of normal child, 18
Werdnig-Hoffmann paralysis, 307
—— —— clinical picture in, 307
—— —— duration of, 308
—— —— etiology and pathology of, 308
—— —— treatment of, 309
Wet nurse for premature infants, importance of, 56
Wheatena, 91, 142
Whitfield's ointment, 377
Whitlow, 378
Whoop, inspiratory, in bronchiectasis, 215
Whooping-cough, 397
—— age- and seasonal incidence of, 398
—— albuminuria in, 253
—— as cause of anorexia, 355
—— —— of vomiting, 358
—— bacteriology and pathology of, 398
—— clinical picture in, 398
—— complicated by broncho-pneumonia, 200, 203, 400
Whooping-cough, complicated by epistaxis, 400
—— convulsions in, 349, 400
—— incubation period of, 398
—— influence of, on tuberculosis, 424
—— leading to bronchiectasis, 214
—— mortality of, 33, 389
—— onset of, 398
—— period of infectivity in, 390
—— prognosis of, 400
—— quarantine period and rules to be observed in, 390
—— recurrences of, 399
—— return to school after exposure to infection of, 390
—— stages of, 398
—— treatment of, 400
—— wasting due to, 400
Widal test, 406
—— —— in paratyphoid fever, 410
—— —— lymphadenoma and, 246
—— —— miliary tuberculosis and, 425
Wind, a symptom of underfeeding, 75
—— as cause of colic, 28, 76, 357
—— breaking of, in infant-feeding, 73, 142, 143
—— swallowing, 73, 75
—— —— (*see also* Aerophagia)
Winter milk deficient in vitamin D, 102
Witch-hazel ointment, 138
Worms, intestinal, 472
Woulfe's bottle, 204, 213
Write, ability to, 14
Wry-neck following hæmatoma of sternomastoid, 11, 46

X

X-ray examination in aerophagia, 142
—— —— in appendicitis, 175
—— —— in asthma, 221
—— —— in bronchiectasis, 216, 217
—— —— in calculus, 254
—— —— in cardiac dilatation, 226
—— —— in cardiospasm, 161
—— —— in cœliac disease, 110

X-ray examination in congenital atelectasis of lung, 52
—— —— —— dislocation of hip, 362
—— —— —— pyloric stenosis, 166
—— —— —— syphilis of bones, 480
—— —— in enlargement of thymus, 218
—— —— in hæmaturia, 254
—— —— in hydrocephalus, 332
—— —— in obstruction of œsophagus and stomach, 358
—— —— in osteogenesis imperfecta, 368
—— —— in rheumatoid arthritis, 459, 460
—— —— in rickets, 104
—— —— in scurvy, 108
—— —— in tuberculosis, 419
—— —— —— miliary, 425
X-ray examination in tubercolosis of hip, 437
—— —— —— pulmonary, 422
—— —— in tuberculous pleurisy, 435, 436
—— treatment of laryngeal papilloma, 194
—— —— of lymphadenoma, 247
—— —— of ringworm, 377
—— —— of spleno - medullary leukæmia, 242
—— —— of warts, 379
—— —— of whooping-cough, 401

Y

Y-dysentery, 116, 118
Yeast as source of vitamin B, 105
—— Fleischmann's, 105
Yellow atrophy, acute or subacute, 184

Z

Zwieback, 92, 93

PRINTED AT
THE CHAPEL RIVER PRESS,
KINGSTON, SURREY.
F30.830.